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THE PLANT DISEASE REPORTER

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PLANT DISEASE EPIDEMICS
and
IDENTIFICATION SECTION

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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Plant Disease Epidemics and Identification Section serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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PLANT DISEASE EPIDEMICS AND IDENTIFICATION SECTION

Horticultural Crops Research Branch — Plant Industry Station, Beltsville, Maryland
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STUDIES OF THE HOST RANGE OF MELOIDOGYNE HAPLA[1]

Timothy A. Gaskin and H. W. Crittenden[2]

This report covers studies of the host range of Meloidogyne hapla Chitwood 1949 conducted in the field and greenhouse at Newark, Delaware.

Earlier work by Hare (1) on pepper and by Sasser (2) on a variety of crops demonstrated that M. hapla has a wide host range. This study is an extension of their work.

Methods

Soil heavily infested with M. hapla was used in pots placed in the greenhouse and in cement-lined pits at the University Farm. This soil was prepared by mixing with it chopped, heavily galled tomato roots. The test plants were grown in this soil. All of the plants were grown from seed[3], except strawberry and potato. After 30 or more days (usually 45 days), the roots were examined and scored. The nematodes were identified by the perineal pattern and type of gall produced.

Results

In interpreting the results reported in Table 1 several factors should be kept in mind. A plant was considered susceptible when adult females and eggs were found on the roots. The numbers used, e. g. 5/10, indicates the number of plants found infected, 5, compared with number tested, 10. The ratings, modified somewhat from Sasser (2), are: none -- no infection; light -- few adult females; moderate -- a moderate number of galls and adult females; and severe -- a high percentage of roots infected with nematodes.

Discussion

The results of these tests indicate that M. hapla has a wide host range. This fact must be taken into consideration when attempting a control program based on rotations.

Another interesting and important point is the high resistance of the plants tested in the Gramineae.

Table 1. Relative susceptibility of plant species to Meloidogyne hapla.

Scientific Name	Common Name	Horticultural Variety	Susceptibility Rating
Gramineae			
Avena sativa L.	Oat	Lee	0/20 None
Hordeum vulgare L.	Barley	Wong	0/20 None
Secale cereale L.	Rye	Imperial	0/20 None
Sorghum vulgare Pers.	Sudan grass	*	0/20 None
Triticum aestivum L.	Wheat	Thorne	0/20 None
		Nured	0/20 None
		Seneca	0/20 None

[1]Published as Miscellaneous Paper No. 241 with the approval of the Director of the Delaware Agricultural Experiment Station. Contribution No. 80 of the Department of Plant Pathology.

[2]Formerly Graduate Research Assistant and Assistant Research Professor, respectively.

[3] The writers wish to thank the Associated Seed Growers Inc., New Haven, Connecticut, for supplying much of the seed.

Table 1. cont'd.

Scientific Name	Common Name	Horticultural Variety	Susceptibility Rating
Zea Mays L.	Sweet Corn	Cheddarcross	0/20 None
		Carmelcross	0/20 None
		Golden Cross Bantam	0/20 None
		Marcross	0/20 None
		Golden Beauty	0/20 None
		Golden Jewel	0/20 None
		Spancross	0/20 None
		Bouquet	0/20 None
		Iochief	0/20 None
		Cannearly	0/20 None
		Hoosier Gold	0/20 None
	Pop corn	Golden Pearl	0/20 None
Liliaceae			
Asparagus officinalis L.	Asparagus	Martha Washington	0/5 None
Allium Cepa L.	Onion	Yellow Globe Danvers	17/20 Mod.
A. Porrum L.	Leek	Swiss Special	18/20 Mod.
Chenopodiaceae			
Beta vulgaris L.	Beet	Early Blood Turnip	5/20 Light
	Chard	Lucillus	7/20 Light
		Rubcoa	10/20 Light
Chenopodium album L.	Pigweed		5/8 Mod.
Rosaceae			
(X Fragaria ananassa Du.)	Strawberry	*	20/20 Severe
Leguminosae			
Medicago sativa L.	Alfalfa	Williamsburg	20/20 Severe
Phaseolus coccineus L.	Scarlet runner bean		16/20 Mod.
P. limensis Macf.	Lima bean	Fordhook U.S.242	13/20 Mod.
		N.R.B. U.S.253	9/20 Light
		N.R.B. U.S.151	15/20 Mod.
P. vulgaris L.	Common bean	Wade	16/20 Mod.
Pisum sativum L.	Pea	Premium Gem	3/7 Mod.
Trifolium pratense L.	Red clover	*	20/20 Severe
Arachis hypogaea L.	Peanut	N C-2	38/45 Severe
Vicia Faba L.	Broad bean	Broad Improved Long Pod	4/9 Light

Table 1. cont'd.

Scientific Name	Common Name	Horticultural Variety	Susceptibility Rating
Glycine Max (L.) Merr.	Soybean	Adams	5/5 Severe
		Anderson	1/5 Light
		Blackhawk	5/5 Severe
		Laredo	1/5 Light
		Lincoln	5/5 Mod.
		Mukden	5/5 Severe
		Monroe	5/5 Severe
		Virginia	5/5 Mod.
Euphorbiaceae			
Ricinus communis L.	Castor bean	Red Spire	5/10 Light
Malvaceae			
Hibiscus esculentus (L.) Moench.	Okra	Emerald	0/20 None
Umbelliferae			
Apium graveolens L.	Celeriac	Large Prague	14/20 Mod.
Daucus Carota L.	Carrot	Red Core Chantenay	5/9 Light
Anethum graveolens L.	Dill	Long Island Mammoth	10/10 Severe
Foeniculum vulgare Mill.	Florence Fennel	Giant Early	7/10 Mod.
Pastinaca sativa L.	Parsnip	Guernsey	7/10 Mod.
Convolvulaceae			
Ipomoea purpurea (L.) Roth.	Morning glory		12/20 Mod.
Valerianaceae			
Valerianella olitoria Poll.	Corn salad		5/20 Light
Cruciferae			
Brassica juncea (L.) Coss.	Mustard	Chinese Broadleaf	10/10 Severe
		Florida Broadleaf	10/10 Severe
		Fordhook Fancy	10/10 Severe
		Southern Giant Curled Long Standing	10/10 Severe
B. perviridis L.	Mustard	Tendergreen	10/10 Severe
B. pekinensis (Lour.) Rupr.	Chinese Cabbage	Wong Bok	7/10 Light
		Chihli	5/10 Light
B. fimbriata D. C.	Kale	Tall Green Curled Scotch	10/10 Severe
		Dwarf Siberian (Sprouts)	5/10 Light
		Dwarf Green Scotch	5/15 Mod.
		Dwarf Blue Scotch	11/15 Mod.

Table 1. cont'd.

Scientific Name	Common Name	Horticultural Variety	Susceptibility Rating
B. caulorapa Pasq.	Kohlrabi	Purple Vienna	15/15 Mod.
		White Vienna	10/15 Light
B. Rapa L.	Turnip	Cowhorn	11/20 Light
		Amber Globe	10/15 Mod.
		Golden Ball	15/15 Mod.
		Pomeranian White Globe	7/15 Light
		Purple Top Milan	11/15 Mod.
		Purple Top Strap	13/15 Mod.
		Purple Top White	10/15 Mod.
		Shogoin	7/10 Light
		White Flat Dutch	7/15 Light
		White Milan	13/15 Severe
		Yellow Aberdeen, Purple Top	11/19 Mod.
B. septiceps L.		Seven Top	7/10 Light
B. napobrassica (L.) Mill.	Rutabaga	Sweet Russian	9/10 Light
		American Purple Top	5/10 Light
B. oleracea var. acephala D. C.	Collard	Vates	7/10 Light
		Georgia	5/10 Light
		Morris Heading	4/10 Light
B. oleracea var. botrytis L.	Cauliflower	Snowball	7/10 Light
		Super-snowball	9/10 Light
B. oleracea var. capitata L.	Cabbage	Badger Market	3/10 Light
		Casi	7/10 Light
		Canadian Acre	3/5 Mod.
		Charleston Wakefield	5/7 Mod.
		Copenhagen Market	3/5 Light
		Danish Ballhead	5/10 Light
		Early Glory	7/10 Mod.
		Early Round Dutch	9/10 Mod.
		Glory of Enkhuizen	6/10 Light
		Globe	7/10 Light
		Golden Acre	3/5 Light
		Green Acre	7/10 Mod.
		Jersey Queen	3/5 Mod.
		Jersey Wakefield	8/10 Light
		Red Danish	10/10 Mod.
		Marion Market	9/10 Mod.
		Midseason Market	7/10 Mod.
		Penn State Ball Head	3/10 Light
		Penn Valley	5/10 Mod.
		Premium Late Flat Dutch	3/5 Mod.
		Red Acre	7/10 Light
		Resistant Detroit	5/10 Light
		Savoy, Perfection Drumhead	8/10 Light
		Stein's Flat Dutch	7/10 Light
		Wisconsin Ball Head	4/5 Mod.
		Wisconsin All Seasons	3/10 Light
		Wisconsin Hollander	4/10 Mod.
B. oleracea var. gemmifera Zenk.	Brussels Sprouts	Long Island Improved	10/10 Mod.

Table 1. cont'd.

Scientific Name	Common Name	Horticultural Variety	Susceptibility Rating
B. oleracea var. italica L.	Broccoli	DeCicco	5/10 Light
		Grand Central	4/10 Light
		K & V	7/10 Light
		Late	7/10 Light
		Medium	9/10 Light
		Midway	7/10 Light
		Texas 107	7/8 Mod.
Raphanus sativus L.	Radish	Brightest Long Scarlet	7/10 Light
		Buccaneer	10/10 Severe
		Cavalier	10/10 Light
		Celestial	9/10 Mod.
		Chartier	7/10 Mod.
		Cherry Belle	9/10 Light
		Chinese Rose Winter	6/10 Light
		Cincinnati Market	3/10 Light
		Comet	10/10 Mod.
		Crimson Giant	7/10 Mod.
		Early Scarlet Globe, Med. Top	3/5 Mod.
		Early Scarlet Globe, Rice's Med. Top	3/5 Mod.
		Early Scarlet Globe, Short Top	5/5 Mod.
		French Breakfast	7/10 Mod.
		Long Black Spanish	7/10 Light
		Philadelphia White Box	5/7 Mod.
		Round Black Spanish	3/10 Light
		Saxa	7/10 Light
		Southern Market Globe	10/10 Mod.
		Sparkler	8/10 Severe
		White Strasbourg	7/10 Mod.
Lepidium sativum L.	Cress	Extra Fine Curled	10/10 Severe
Solanaceae			
Capsicum frutescens L.	Pepper	World Beater	20/20 Severe
Lycopersicon esculentum Mill.	Tomato	Wiltmaster	20/20 Severe
		Earliana	20/20 Severe
		Marglobe	20/20 Severe
		Ponderosa	20/20 Severe
		Rutgers	20/20 Severe
		Yellow Pear	20/20 Severe
		Red Cherry	20/20 Severe
		Potato-leafed	20/20 Severe
Solanum melongena L.	Eggplant	Black Beauty	20/20 Severe
S. tuberosum L.	Potato		20/20 Mod.
Polygonaceae			
Rumex Acetosa L.	Sorrel	Narrow-leaved	4/20 Light
Plantaginaceae			
Plantago lanceolata L.	Ribwort		5/9 Mod.
P. major L.	Common plantain		3/9 Light

Table 1. cont'd.

Scientific Name	Common Name	Horticultural Variety	Susceptibility Rating
Aizoaceae			
Tetrogonia expansa L.	New Zealand spinach		2/5 Light
Curcurbitaceae			
Citrullus vulgaris Schard.	Watermelon	Charlestown Gray	1/20 Light
		Congo	1/20 Light
		Early Kansas	3/20 Light
		Dixie Queen	0/20 None
		Fairfax	0/20 None
		Florida Giant	0/20 None
		Honey Cream	1/20 Light
		Hoosier Black	0/20 None
		Northland Hybrid	0/20 None
		Rhode Island Red	0/20 None
		Wilt Resistant Ironsides	0/20 None
Curcurbita pepo L.	Pumpkin	Big Tom	3/10 Light
	Squash	Fordhook Zucchini	5/10 Light
C. maxima L.	Squash	Warted Hubbard	10/10 Mod.
Cucumis Melo L.	Cantaloupe	Hale's Best	20/20 Severe
C. sativus L.	Cucumber	Marketer	20/20 Severe
		Santee	10/20 Light
Compositae			
Cichorium Intybus L.	Chicory	Witloff	14/20 Mod.
Helianthus annuus L.	Sulflower		6/10 Light
Lactuca sativa L.	Lettuce	Iceberg	19/20 Severe
Taraxacum officinale Web.	Dandelion	Italian	15/20 Mod.
Tragopogon porrifolium L.	Salsify	Mammoth Sandwich Island	5/10 Light
Tagetes erecta L.	Marigold	African Double Mixed	2/20 Light

*Variety not known

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AN OUTBREAK OF ONION BLOAT IN SOUTHERN NEW YORK

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Abstract

Recent outbreaks of onion bloat, caused by Ditylenchus dipsaci Kühn, in southern New York are attributed to the planting of infected onion sets produced in the Midwest. No infestations due to infected sets occurred in 1955, presumably because of cooperation with the distributors of sets and the inspection of sets shipped to southern New York.

Onion bloat, caused by Ditylenchus dipsaci Kühn, occurred prior to 1940 in the muckland region of Orange County, New York (2). The disease still occurs in a few fields in the vicinity of the original infestation. Until the last few years it was limited to this area. Many new infestations, scattered throughout this entire muckland area, were found in 1954. It is estimated that the fields now affected comprise a total area of approximately 200 acres. By questioning growers it has been determined that a few new infestations were first noted in 1952 and 1953, but the majority were first located in 1954. A few new infestations were found in 1955, but all of these were attributable to contamination from adjacent infested fields.

A survey of the area has indicated that the original infestation reported in 1939 was not the source of the new infestations. Newhall (3) suggested that the original outbreak in Orange County and that near Canastota, New York, resulted from the planting of infected onion sets that were produced outside of the State. Strong circumstantial evidence indicates that the new infestations have resulted from the planting of infected onion sets that were produced in the Midwest. The following facts have led the writer to conclude that infected sets are the cause of the infestations. 1) Practically all newly infested fields had been planted with sets at least once within 3 years prior to the appearance of the disease. 2) Several entire fields that were not known to be infested before 1954 became infested that year following the planting of a crop of midwestern sets. In each instance, the infestation that occurred was uniform over the entire field or the area planted with such sets. This type of infestation is quite different from the usual type, which appears at the time the onions mature as a bare spot that resembles a lightening strike and persists and increases in area from year to year (1). Losses in these fields ran as high as 100 percent. Some of these fields were planted with onions (both seed and sets) again in 1955, and 100 percent losses were incurred in each case. 3) Dr. C. I. Hannon, formerly of this department, and the writer examined samples of onion sets shipped from the Midwest in the fall of 1954; some samples (3 out of 42 examined) contained bulbs infected with D. dipsaci.

In cooperation with the main distributor of onion sets in Orange County, the writer examined subsamples of a large portion of the lots of sets shipped to the county in the spring of 1955. No infected bulbs were found in any of these samples. No new infestations were found in 1955 except those that could be attributed to the use of machinery and implements previously used in infested fields or, as in one case, to the use of contaminated crates.

Three severe windstorms resulted in a widespread movement of soil in a major portion of the Orange County muckland during the spring of 1955. Two extensive floods occurred during the harvest period. Subsequent infestations will be carefully investigated in an attempt to determine whether wind and flooding are important factors in the movement of D. dipsaci from one field to another.

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1. Newhall, A. G. 1939. Notes on onion diseases in New York. Plant Dis. Reptr. 23: 229-231.
2. Newhall, A. G., et al. 1939. A survey of the occurrence of the bulb or stem nematode on onions in the State of New York. Plant Dis. Reptr. 23: 291-293.
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REACTION OF CUCUMBER VARIETIES TO FIVE ROOT-KNOT NEMATODES (MELOIDOGYNE SPP.)

N. N. Winstead and J. N. Sasser

SUMMARY

Fifty cucumber varieties, breeding lines, or plant introductions and two varieties of Cucumis anguria were tested for resistance to five root-knot nematodes, Meloidogyne incognita, M. incognita acrita, M. javanica, M. arenaria, and M. hapla. All cucumber varieties tested were resistant to M. hapla, and susceptible to the other four root-knot nematodes. The two Gherkin varieties differed in their susceptibility to different root-knot species. Plants of the Small Gherkin variety were susceptible only to M. incognita and M. arenaria, while those of the West India Gherkin were susceptible only to M. incognita acrita.

Root-knot caused by nematodes of the genus Meloidogyne, Goeldi, 1887, was first reported as a plant disease by Berkeley (1) in 1855. The host plant was cucumber, Cucumis sativus L., growing in an English greenhouse. Generally, cucumber has been regarded as one of the most susceptible crops to root-knot (6). This disease has frequently been severe in North Carolina on late spring and fall cucumber crops. Few reports of the reactions of cucumber varieties to Meloidogyne spp. are found in the literature. Recently Sasser (5) reported that plants of Marketer cucumber variety were susceptible to four species and that A & C, Cubit, and Marketer varieties were resistant to M. hapla. Dropkin (4) reported that the Long Green cucumber variety was very susceptible to M. incognita acrita.

In this investigation 50 varieties, breeding lines or P. I. lines of cucumber and two varieties of C. anguria L. were tested to determine their reactions to the five Meloidogyne species and subspecies collected in North Carolina. These were: Meloidogyne incognita (Kofoid and White, 1919) Chitwood, 1949; M. incognita acrita Chitwood, 1949; M. hapla Chitwood, 1949; M. arenaria (Neal, 1889) Chitwood, 1949; and M. javanica (Treub, 1885) Chitwood, 1949.

Materials and Methods

Inoculum was increased from single egg mass cultures on Rutgers tomato plants using techniques previously described (5). Infested soil containing tomato roots bearing egg masses was used as inoculum. In preliminary tests seeds were planted directly into pots containing inoculum. In such cases seedlings were severely galled and frequently died. To avoid this in subsequent tests, seeds were planted in steam sterilized soil in 2 1/2-inch clay pots and allowed to grow from 2 to 3 weeks to establish good root systems. They were then transplanted, one plant per pot, to 4-inch clay pots. Four pots of each variety were tested in each of two tests.

Root-knot ratings were made 2 months after inoculation by washing the soil from the roots and examining macroscopically. The following rating system was used: 0 -- no infection, or if larvae entered the roots they did not develop into egg-laying females; 1 -- very light infection, or only an occasional female egg mass; 2 -- light infection with mature females and a few egg masses easily seen with the naked eye; 3 -- moderate infection with mature females and egg masses moderately abundant; and 4 -- severe infection with mature females and egg masses very abundant. A dissecting microscope was used in making the 0 or 1 class determinations. Varieties falling into the 0 to 2 classes are considered resistant.

Experimental Results

The varieties tested and their reactions to five root-knot nematodes are presented in Table 1. Little or no differences were observed in the reactions of plants of the same varieties from different sources. All varieties of cucumber tested were susceptible to M. incognita acrita, M. incognita, M. javanica, and M. arenaria and all these varieties were resistant to M. hapla. However, resistance to each of the root-knot nematodes tested was found within the two varieties of C. anguria. Small Gherkin was resistant to M. hapla, M. javanica, and M. incognita acrita, while West India Gherkin was susceptible only to M. incognita acrita. Very large galls (except with M. hapla) frequently formed on the older roots of these resistant plants;

however, very few galls were observed on the secondary roots. When galls were examined numerous nematodes were found but egg masses were seen infrequently.

Table 1. Reaction of cucumber varieties and lines to five root-knot nematodes (Meloidogyne spp.).

Variety or Line	Sources of seed[a]	Susceptibility Ratings				
		M.incognita	M.incognita acrita	M.hapla	M.javanica	M.arenaria
Cucumis sativus						
A and C	A. F.	4	4	1	4	4
Ashley	A.	4	4	1	4	4
Black Diamond	F.	4	4	1	4	4
Boston Pickling	F.	4	4	1	4	4
Cambridge F_1 Hybrid	A.	4	4	0	4	4
Chicago Pickling	A. F.	4	4	2	4	3
Colorado	A.	4	4	1	4	4
Cubit	A. F.	4	4	1	4	3
Davis Blend	F.	4	4	1	4	3
Earliest of All	A. F.	4	4	2	4	3
Early Cluster	F.	4	4	1	4	4
Early Fortune	A. F.	4	4	1	4	4
Early Russian	A.	3	4	1	4	4
Improved Long Green	A. F.	4	4	2	4	3
Japanese Climbing	A.	4	4	1	4	3
Lemon	A. F.	4	4	1	4	3
Longfellow	A. F.	4	4	1	4	3
Long Stays Green	A.	4	4	1	4	4
Maine No. 2	A.	4	4	1	4	4
Marketer	A. F.	4	4	1	4	4
Model	A.	4	4	1	4	4
National Association Pickling	A.	4	4	1	4	4

Table 1. Cont.

Variety or Line	Sources of seed[a]	Susceptibility Ratings				
		M.incognita	M.incognita acrita	M.hapla	M.javanica	M.arenaria
National Pickling	F.	4	4	1	4	4
New York	A.	3	3	2	4	4
Niagara	A.	3	3	2	4	4
Ohio MR 17	A.	4	3	2	4	4
Packer	A.	3	3	2	4	4
Palmetto	A.B.F.	4	4	1	4	4
Puerto Rico 39	A. K.	4	4	1	4	4
Santee	A. B.	4	4	2	4	4
Smoothie	J.	4	4	1	4	4
Snows Perfection Pickling	A.	4	4	2	4	4
Stays Green	A.	3	4	1	4	4
Stono	A.	4	4	1	4	4
Straight Eight	A. F.	3	4	2	4	4
White Wonder	A.	3	4	2	4	4
Wisconsin SR 6	A. W.	4	4	2	3	4
Wisconsin SR 10	A. W.	4	4	1	3	4
Wisconsin SMR 12	A.	4	3	1	4	4
Wisconsin SMR 12-9	W.	3	3	1	4	3
Wisconsin SMR 12-12	W.	4	3	1	4	4
York State	A.	4	3	1	4	4
S. C. 849A-2	B.	4	3	1	4	4
N. C. 1955-114	N.	4	4	0	3	4
N. C. 1955-115	N.	4	4	1	3	3
N. C. 1955-139	N.	4	4	1	4	3
N. C. 1955-146	N.	4	4	0	3	3

Table 1. Cont.

Variety or Line	Sources of seed[a]	Susceptibility Ratings				
		M.incognita	M.incognita acrita	M.hapla	M.javanica	M.arenaria
P. I. 173889[b]	B. J.	4	4	2	4	3
P. I. 175111	B. J.	4	3	1	4	4
P. I. 175120	B. J.	4	3	1	4	4
P. I. 179676	B. J.	4	4	1	4	3
Cucumis anguria						
Small Gherkin	F.	3	2	1	2	4
West India Gherkin	A.	2	3	1	1	2

[a]A -- Associated Seed Growers, Inc.; F -- Ferry Morse Seed Co.; K -- The Kilgore Seed Co.; B -- Dr. W. C. Barnes, Charleston, South Carolina; J -- Dr. Edwin James, Experiment, Georgia; W -- Dr. J. C. Walker, Madison, Wisconsin; N -- W. S. Barham and N. N. Winstead, Raleigh, North Carolina; M -- Dr. Mitchell Jenkins, Castle Hayne, North Carolina.
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NEW HOST PLANTS OF PLANT PARASITIC NEMATODES IN CALIFORNIA

Robert B. Nesbitt[1]

During November of 1955, the citrus-root nematode, Tylenchulus semipenetrans Cobb (4), was found in Orange County, California attached to field grown persimmon roots, including Diospyros lotus seedlings and D. lotus rootstock with scions of the Hachiya variety.

Washed root samples and associated soil samples from the persimmon gave high populations of citrus nematode larvae and males collected by means of the Baermann funnel method. Direct examination of stained (acid fuchsin) fibrous persimmon roots disclosed the presence of attached mature females and egg masses.

Several members of the Rutaceae family (2, 3) and olive varieties appear to have been the the only hosts of Tylenchulus semipenetrans on record at the time of these findings.

In March, 1955, the root lesion nematode, Pratylenchus scribneri Steiner (1), was found in the roots of roses which were being grown for the cut flower market.[2] Earlier records show this nematode to have been obtained in other States from potato tubers and amaryllis roots.

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[1] Deputy County Agricultural Commissioner and Plant Pathologist, Orange County Department of Agriculture, Orange County, California.

[2] The writer wishes to thank Drs. M. M. Allen of the University of California at Berkeley, S. Sher of the University of California at Riverside, and W. Hart of the California State Bureau of Plant Pathology for their collaboration regarding the finding of Pratylenchus scribneri and W. Hart regarding the Tylenchulus semipenetrans on persimmon roots.

NEMATODES ASSOCIATED WITH ROSES IN A SURVEY OF COMMERCIAL GREENHOUSES[1]

A. F. Schindler[2]

In an attempt to determine the cause of what seemed to be a nematode problem in commercial greenhouses growing roses, it was advisable to determine whether there was a correlation between the disease symptoms and the genera and quantity of nematodes associated therewith. Therefore, a survey was conducted in which 126 soil samples from 34 localities (i. e. growers) in 14 States were examined for nematodes.

In general, samples were taken from areas of greenhouse benches which contained plants showing chlorosis and poor growth. Approximately 2 pounds of soil, usually including the roots of a rose plant, were obtained for each sample. Each sample was processed by roiling it in a bucket of water and then running the water through 30-, 100-, and 200-mesh screens. The washings from the last two screens were then examined for nematodes. The modified Baermann funnel technique as described by Christie and Perry (1) was not used in this study since it was found that large numbers of dagger nematodes (Xiphinema spp.) would settle on the surface of the screen debris and remain there instead of coming down through the funnel. The possibility of similar behavior should be kept in mind when one is working with large forms of nematodes in soil high in rotting organic matter such as that found in greenhouse rose beds.

Quantitative data are presented for only genera considered to be plant parasitic (Table 1). Other, free-living genera found were Acrobeloides, Alaimus, Aporcelaimus, Aphelenchus, Butlerius, Cephalobus, Diplogaster, Diplogasteroides, Diploscapter, Dorylaimus, Eucephalobus, Labronema, Monhystera, Mononchus, Nygolaimus, Odontopharynx, Panagrolaimus, Prismatolaimus, Rhabditis, Sectonema, and Tripyla.

In all instances where large numbers of plant parasitic nematodes were found plants showed poor growth. In almost all of these cases Xiphinema species predominated. From the data obtained (Table1) it appears that both Xiphinema and Pratylenchus are widely distributed in greenhouse rose beds of the States surveyed. Expressed in percentage of the total number of samples examined, both genera were also found to occur more frequently than any of the others. The dagger nematodes, however, were found in moderate to heavy infestations in 60 percent of the samples in which they occurred, approximately twice as often as the meadow nematodes (Pratylenchus). It is felt that these two genera present a problem in commercial rose-growing greenhouses. One species of Xiphinema (X. diversicaudatum) has already been shown by experiments to cause galls on rose roots, which at first glance can easily be mistaken for root knot (2). Additional experiments are now in progress to show the effects of this species on Manetti rose plants.

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[1] Scientific Article No. A547, Contribution No. 2688 of the University of Maryland Agricultural Experiment Station, Department of Botany. This research was supported in part by a research grant from Roses, Incorporated, to the University of Maryland in collaboration with the Nematology Section, Agricultural Research Service, Beltsville, Maryland.

[2] Now Assistant Nematologist, United States Department of Agriculture, Agricultural Research Service, Horticultural Crops Research Branch, Beltsville, Maryland.

Table 1. Analysis of the genera of plant parasitic nematodes found in soil samples from commercial, rose-growing greenhouses of 14 States.

Nematode genera	California	Colorado	Connecticut	Illinois	Indiana	Kentucky	Massachusetts	Michigan	New Hampshire	New Jersey	New York Long Island	Pennsylvania	South Carolina	Virginia	Total No.	Percent of total number samples examined	Number with moderate to heavy infestations
Number of growers	1	1	2	1	2	1	1	1	1	6	9	6	1	1	34		
Number of samples	10	2	23	2	4	2	2	2	1	25	19	27	4	3	126		
Xiphinema		1	7	2	4			1	1	2	14	19	2		53	42.1	32
Pratylenchus	1	2	7							12	14	13	4	3	56	44.4	16
Criconemoides										2	1	5			8	6.4	5
Paratylenchus	2									6		3			11	8.7	2
Helicotylenchus								2		2	1	1			6	4.8	2
Hemicycliophora												2			2	1.6	1
Belonolaimus			1												1	0.8	1
Trichodorus			1												1	0.8	1
Tylenchus			3			1					1	1			6	4.8	1
Aphelenchoides			6								2	2			10	8.0	0
Psilenchus			1					1		2					4	3.2	0
Meloidogyne (hapla)												1			1	0.8	0

CUCUMBER MOSAIC VIRUS AND ALFALFA MOSAIC VIRUS ISOLATED FROM DAPHNE ODORA[1]

J. A. Milbrath and Roy A. Young

SUMMARY

Strains of cucumber mosaic virus were isolated from all of 45 Daphne odora plants tested regardless of whether the plants appeared normal or diseased. Alfalfa mosaic virus was also present in at least 15 of the 45 plants.

The viruses were identified on the basis of reaction in cucumber, bountiful bean, broad bean, and cowpea and cross-protection tests with ATC #10 of CMV and with an Oregon State type culture of AMV.

A mosaic disease of Daphne odora Thunb. was reported from New Zealand in 1941 by Chamberlain and Matthews (4), but the causal virus was not classified. A similar disease of D. mezereum L. has been reported subsequently from Holland (6), England (2, 5), Denmark (1) and New Zealand (2). Cucumber mosaic virus has been listed as the virus causing Daphne mosaic in New Zealand (3) and England (5), but van Katwijk in Holland apparently considered the Daphne mosaic virus a distinct entity (6).

Chamberlain and Matthews (4) described the characteristic symptoms on D. odora as a pale yellowish mottling, of variable pattern, that appeared most clearly on younger leaves. They considered that the disease had no effect on growth since mosaic-infected plants kept under observation for 12 months grew as vigorously and flowered as profusely as healthy plants. The mosaic disease on D. mezereum is similar but is much more severe and results in defoliation and death of affected plants (6). With both D. odora (4) and D. mezereum (6) transmission from affected to healthy Daphne plants was obtained readily by grafting but with difficulty by mechanical inoculation.

In 1952, Federal plant quarantine officials became interested in whether or not the leaf mottling present in many D. odora plantings in the Pacific Northwest might be the Daphne mosaic reported in Europe and New Zealand. Subsequent, studies were initiated at Oregon State College to determine the nature of the mottling observed on Daphne odora plants.

Isolation of the Causal Viruses

Cucumber mosaic was recovered from a small potted D. odora plant which had been selected for study because of its mottled yellowed leaves. Six cucumber plants were inoculated mechanically, using carborundum and juice extracted in 0.2 phosphate buffer from the young unfolding tip leaves of Daphne. The plants were inoculated when the cotyledons were almost fully expanded. Four days after inoculation, 3 to 6 distinct chlorotic local lesions about 1/8 to 1/4 inch in diameter appeared on the inoculated cotyledons. Seven to 9 days after inoculation 5 of the 6 plants showed a strong mosaic mottle which started as a yellow vein mottle on the expanding primary leaf. Subsequent leaves also showed a similar mottle.

Cucumbers were next inoculated with juice from the young tip leaves from 17 different D. odora and 2 D. cneorum L. plants from 10 different properties. Cucumber mosaic was recovered from all 17 D. odora plants but not from the D. cneorum.

A number of commercial and home plantings of Daphne odora were then examined to determine the extent of mottling and range of symptoms. The following symptom types were observed:

1. Normal D. odora with unmottled foliage (Fig. 1A)
2. D. odora var. marginata with normal white marginal variegation
3. Mild green mottling
4. Yellow-green mottling (Fig. 1 B)
5. Dwarf plants with severe yellow-green mottle
6. Plants with mottled foliage and shortened internodes resulting in a rosette effect.

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[1]Technical Paper No. 957, Oregon Agricultural Experiment Station.

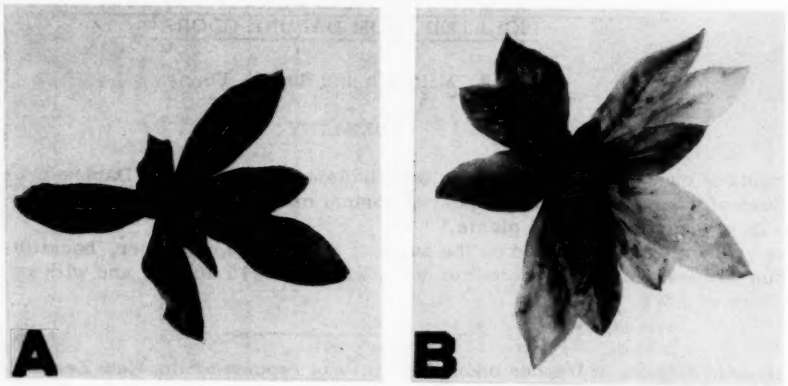

FIGURE 1. Tips of two of the Daphne odora plants from which CMV and AMV were recovered. A. Leaves normal green color and no indication of virus; B. Leaves with a yellow green mottling.

FIGURE 2. Variations in local lesions produced by Daphne isolates on cucumber. A. CMV type of local lesion; B. AMV type; C. Mixture of CMV and AMV; D. Mottled symptom of CMV; E. Spreading necrosis of a CMV isolate.

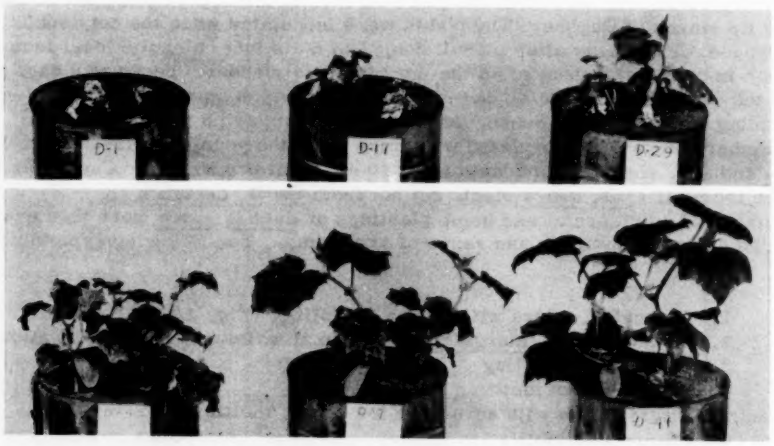

FIGURE 3. Variations in systemic symptoms produced by Daphne isolates in cucumber showing range of severity.

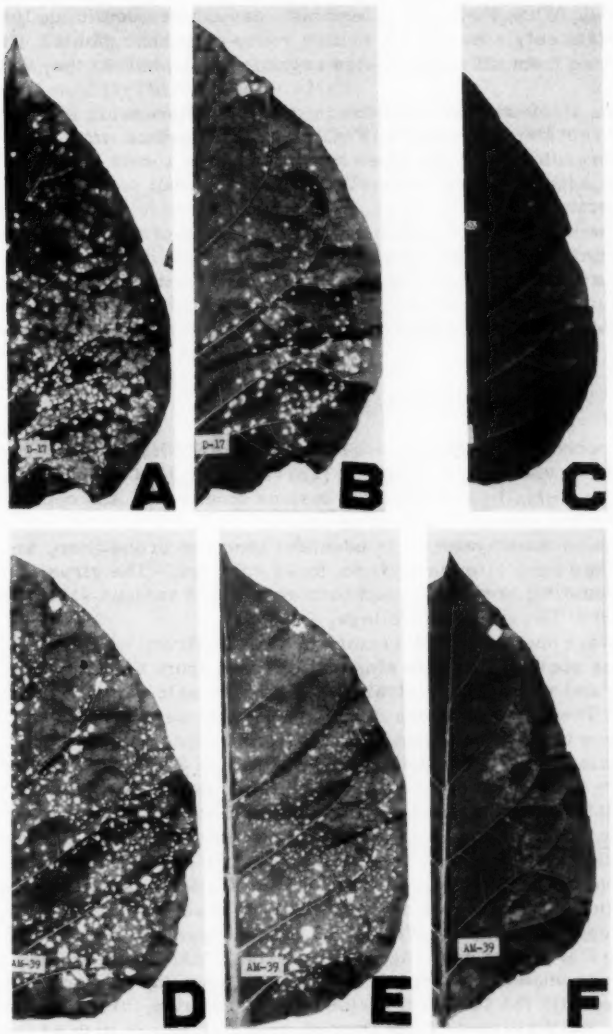

FIGURE 4. Cross protection comparisons of Daphne isolates with type cultures of cucumber mosaic and alfalfa mosaic viruses.

A, B, C. Challenge virus; CMV from Daphne; Established virus: A. None; B. AMV (OSC Type 39); C. CMV (AMTC #10).

D, E, F. Challenge virus AMV (OSC Type 39); Established virus: D. None; E. CMV (AMTC #10); F. Daphne isolate of AMV.

From the observed plants 28 were selected, including two or more plants from each of the above symptom groups. When their young terminal leaves were unfolding, juice was extracted and used to inoculate cotyledons of six to nine young cucumber plants. Cucumber mosaic virus was recovered from all plants tested regardless of whether they appeared normal or diseased.

During these isolation studies great variation in symptom expression was noted on cucumbers inoculated with different Daphne isolates (Fig. 2). Local lesions were classified into four groups: (A) large spreading chlorotic types tending to form zonate rings, (B) smaller discrete pale green spots, (C) chlorotic spots similar to type A but smaller, (D) diffused chlorotic areas without distinct margins.

Systemic symptoms were also very variable (Fig. 3). Some of the isolates were lethal and difficult to maintain on small cucumbers. Others caused a severe mosaic of the foliage and dwarfing of the plants or a distinct mosaic pattern of the leaves without severe reduction in growth. Some of the cucumbers exhibited strong secondary symptoms within a week after inoculation, while others did not show secondary symptoms until three or four secondary leaves had developed.

Identification of the Viruses

The isolated viruses were transferred to a series of cucumbers, bountiful bean, broad bean (Vicia faba L.) and to cowpea (Vigna sinensis (Torner) Savi.). CMV does not infect bountiful bean, produces only small pin point local lesions on cowpea, and occasionally causes local lesions on broad bean but seldom becomes systemic.

Several of the isolates became systemic in bountiful bean and broad bean, and subsequent studies showed that CMV had been eliminated from these cultures. The virus isolated from the secondary growth of bountiful bean and broad bean resembled various strains of alfalfa mosaic virus being studied at Oregon State College.

The Daphne isolates were compared with cucumber mosaic virus, cucumber green mottle mosaic virus, tobacco ring spot, and tomato ring spot viruses from the American Type Culture Collection of viruses and with various strains of alfalfa mosaic virus in the Oregon State College virus collection. These comparisons demonstrated that several strains of CMV and also several distinct strains of AMV had been isolated from Daphne.

Cross-protection studies were attempted using Golden Gem Zinnia and the indicator strain of CMV (ATC #12), but the culture received failed to produce local lesions on zinnias. Two of the CMV isolates from Daphne had produced distinct necrotic lesions on a hybrid tobacco (self-fertile Nicotiana tabacum x N. glutinosa), and some of the strains of alfalfa mosaic in the Oregon State virus collection also produce distinct local lesions on this host. Since CMV and AMV both become fairly uniformly systemic in this tobacco if plants are allowed to develop 10 to 12 leaves after inoculation, this afforded an opportunity to make cross-protection tests using the type cultures as reference materials. When ATC #10 of cucumber mosaic and the Daphne viruses suspected of being CMV were challenged with the two local lesion strains of the Daphne isolates, all gave good cross protection (Fig. 4). However, when the isolates suspected of being AMV were challenged with the two local lesion Daphne isolates, cross protection could not be detected. When the AMV suspects from Daphne were challenged with an Oregon State College type culture of AMV, they gave cross protection, but neither the cucumber isolates from Daphne nor the CMV type cultures gave cross protection.

Discussion

Cucumber mosaic virus was isolated from all of the 45 Daphne odora plants tested in these studies. Forty-four of these plants were obtained from commercial nurseries or home plantings in various parts of Oregon, and one plant from Richmond, Virginia, was selected by F. R. Freund, Supervisor, Crop Pest Inspection, Division of Plant Industry, as a possible virus-free plant because the plant appeared unusually green and healthy. Alfalfa mosaic virus was also present in at least 15 of the Daphne collected. Since D. odora plants are propagated vegetatively and most of the plants used in ornamental plantings in the United States are propagated in the Pacific Northwest, it is likely that these viruses occur generally in D. odora.

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APPLE-GRAIN AND ENGLISH GRAIN APHIDS AS VECTORS OF THE WASHINGTON STRAIN OF THE CEREAL YELLOW-DWARF VIRUS

H. V. Toko and G. W. Bruehl

Of the five aphid species reported as vectors of cereal yellow-dwarf virus in California[1], three have been tried in Washington and found to be effective vectors. They were the apple-grain aphid (Rhopalosiphum fitchii Fitch), the English grain aphid (Macrosiphum granarium Kirby), and the green bug (Toxoptera graminum Rondani). The apple-grain and English grain aphids were reported efficient vectors[1] in California, and they are the most common aphids affecting small grains in Washington[2]. Observations of plants infected by means of the apple-grain and English grain aphids, using a common source of the virus, indicated that the apple-grain aphid not only accomplished a higher percentage of infection but that the disease was more severe in those plants in comparison with those infected by means of the English grain aphid. Trials were conducted to test this observation.

Materials and Methods

The yellow-dwarf virus was transmitted by the apple-grain and English grain aphids in two different stages. The non-viruliferous aphids were allowed to feed on the same source plant and then separated (Fig. 1). Non-viruliferous aphids were taken at birth by the method described by Oswald and Houston[1]. They were reared on healthy cereals until needed. No symptoms of yellow dwarf ever developed on these food plants, even though many aphids were reared on them.

In the first transmission stage Fulghum oats (C.I. 708) were the test plants. In the second transmission Lemhi wheat (C.I. 11415) and white winter barley (C.I. 592) were added to Fulghum oats. These cereals were grown in the greenhouse in 6-inch clay pots, four plants per pot. Temperatures were about 65°-75°F. Water and nutrients were suppled in abundance. Light conditions (March-June, 1955) were good.

In the first transmission non-viruliferous apple-grain and English grain aphids were reared together on a single source plant infected with the Washington strain of cereal yellow-dwarf virus[3]. When they had multiplied, adults of each species were separated and placed on oat seedlings 2 inches tall. Two adults were fed 4 days on each seedling. Apple-grain aphids were placed on 76 seedlings, English grain on 74. No aphids were placed on 48 plants, constituting a check. (Table 1). Each pot was caged separately during the feeding period. The plants were examined to make sure the aphids did not congregate on one plant, or move off the plants. After the 4-day feeding period they were killed by fumigation with nicotine, and periodic fumigations kept them aphid-free thereafter.

The plants infected by means of the apple-grain aphid were distinguishable from those infected by means of the English grain aphid as soon as symptoms became definite, or from about 3 to 4 weeks. At this time the second transmission was begun. A typical pot of oats infected by means of the apple-grain and a representative pot of plants infected by means of the English grain aphid were selected. Again non-viruliferous aphids of each species were placed upon each pot. Each pot was caged alone. After multiplication, the aphids were again separated as to species and placed upon oats, wheat, and barley in the manner outlined above. This second transmission resulted in plants infected with virus transmitted only by the apple-grain aphid, only by means of the English grain aphid, and by both species, each following the other (Fig. 1, at bottom, for final "pedigree" of the virus). In the second transmission stage non-viruliferous aphids of each species were placed upon the cereals, in addition to plants receiving no aphids, as checks. No symptoms were observed on any of these plants.

Results

In the first transmission stage, the apple-grain aphid transmitted cereal yellow-dwarf

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1 Oswald, J. W., and B. R. Houston. 1953. The yellow-dwarf virus disease of cereal crops. Phytopath. 43: 309-313.

2 Johansen, Carl. 1954. Aphids of the State of Washington. Washington Agr. Exp. Sta. Cir. 243.

3 Bruehl, G. W., and H. Toko. 1955. A Washington strain of the cereal yellow-dwarf virus. Plant Dis. Reptr. 39: 547-549.

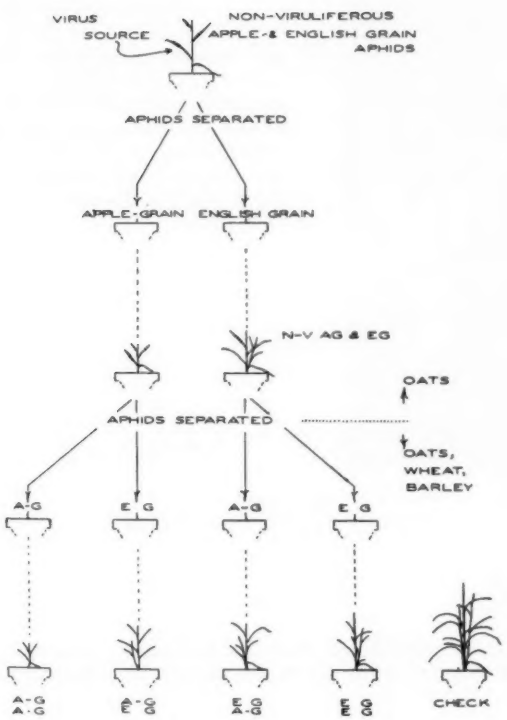

FIGURE 1. Diagramatic presentation of serial transmissions of the cereal yellow-dwarf virus by means of the apple-grain and English grain aphids. The apple-grain - apple-grain transmissions gave the highest percentage of infection and greatest stunting. English grain-English grain and English grain - apple-grain transmissions gave high percentages of infection but moderate disease severity, and apple-grain - English grain gave low percentages of infection.

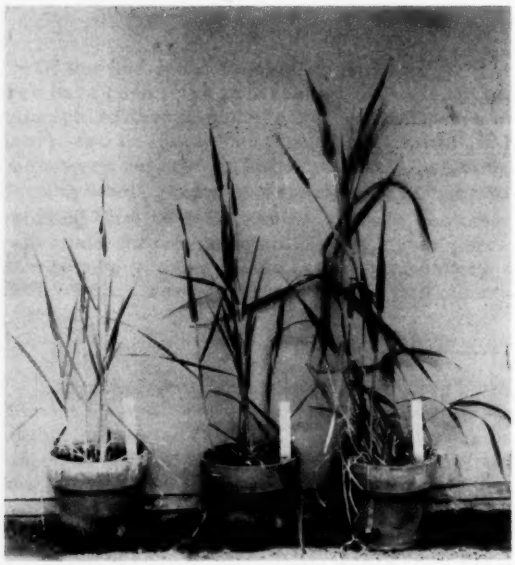

FIGURE 2. Development of cereal yellow dwarf on white winter barley inoculated by means of two species of aphids fed on plants infected by a known vector. Left to right: inoculated by the apple-grain aphid taken from a plant originally infected by the apple-grain aphid; apple-grain aphid in final transmission following transmission by the English grain aphid; healthy plants on right.

Table 1. Summary of the first transmission stage showing the relative efficiency of the apple-grain aphid and the English grain aphid as vectors of the Washington strain of the yellow-dwarf virus.

Vector	Number of plants		Percent transmission	Number of shoots	
	Inoculated	Diseased		Diseased	Healthy
Rhopalosiphum fitchii (Apple-grain aphid)	76	75	98.7	14	4[a]
Macrosiphum granarium (English grain aphid)	74	30	40.5	30	48*
Check	48	0	0	--	87

[a]In the inoculated series all plants or shoots listed as "healthy" exhibited no symptoms of yellow dwarf. Not checked for virus content by aphid recovery.

* Healthy by observation. No symptoms expressed. Virus recovery from these tillers not attempted.

Table 2. Yellow-dwarf virus transmission tests to Fulghum oats, Lemhi wheat, and White Winter barley by use of the English grain aphid and apple-grain aphid from diseased plants previously infected by a known vector.

Original transmission	Second transmission	Number of plants		Percent transmission	Number of tillers[a]		Average weight[b] of plants (gms.)	
		Inoculated	Diseased		Diseased	Healthy	Diseased	Healthy
FULGHUM OATS								
Apple-grain	Apple-grain	22	21	95.4	0	2	1.9	5.2[c]
English grain	Apple-grain	22	18	81.8	11	4	3.4	3.9
Non-viruliferous	Apple-grain	13	0	0	--	13	---	5.0
English grain	English grain	22	20	90.9	20	4	4.5	6.8
Apple-grain	English grain	22	2	9.1	0	20	2.8	5.3
Non-viruliferous	English grain	13	0	0	--	13	---	4.9
Check		46	0	0	--	81	---	6.3
LEMHI WHEAT								
Apple-grain	Apple-grain	15	14	93.3	0	2	1.3	5.2
English grain	Apple-grain	16	10	62.5	2	4	1.2	3.7
Non-viruliferous	Apple-grain	16	0	0	--	23	---	3.2
English grain	English grain	16	7	43.7	7	9	1.8	3.1
Apple-grain	English grain	16	0	0	--	17	---	3.3
Non-viruliferous	English grain	16	0	0	--	16	---	3.1
Check		8	0	0	--	19	---	4.2
WHITE WINTER BARLEY								
Apple-grain	Apple-grain	15	14	93.3	0	2	---	---
English grain	Apple-grain	16	10	62.5	2	4	---	---
Non-viruliferous	Apple-grain	16	0	0	--	23	---	---
English grain	English grain	16	7	43.7	7	9	---	---
Apple-grain	English-grain	16	0	0	--	17	---	---
Non-viruliferous	English grain	16	0	0	--	16	---	---
Check		8	0	0	--	19	---	---

[a] In the inoculated series, all plants or tillers listed as "healthy" exhibited no symptoms of yellow dwarf. Not checked for virus content by aphid recovery.

[b] No weight data were obtained for barley, but from observations of the developing grain the relations of the vector-virus-disease development were the same as in oats and wheat (See Fig. 2B).

[c] Weights of healthy plants varied considerably, as one escape with 3 severely diseased plants had less competition than 4 healthy plants together in a 6-inch pot.

virus to greater numbers of oat seedlings and in more severe form than did the English grain aphid (Table 1). Oat seedlings infected by means of the apple-grain aphid were severely stunted, tillering was sharply reduced (14 tillers on 75 plants), and the leaf discolorations scarlet to purple. Using the English grain aphid, fewer plants were infected, stunting was slight, tillering reduced less (32 tillers on 30 plants), and the discolorations were not so brilliant, being pale orange to light red.

In the second transmission stage, in which plants were infected by means of each aphid species fed upon plants to which the yellow-dwarf virus had been previously transmitted by a known vector, further relationships were revealed (Table 2). Virus transmitted by apple-grain - apple-grain aphids infected over 90 percent of each cereal. Good percentages of infection followed the English grain - English grain transmission and the English grain - apple-grain transmissions, but in apple-grain - English grain only 9 percent of the oat plants and no wheat or barley plants were infected, as judged by absence of visible symptoms. The apple-grain - apple-grain transmitted virus was the most virulent on all three cereals (Fig. 2). The English grain - apple-grain transmitted virus was intermediate in virulence. Too few plants were infected in the apple-grain - English grain series to permit comparison.

Small populations of non-viruliferous aphids of each species produced no visible effects upon plant growth.

A small test using 1, 2, 4, and 8 viruliferous aphids of each species on different oat seedlings showed no "quantitative" differences in transmissions. Disease severity did not increase with number of vectors, nor did 8 English grain aphids bring about the same severity that 1 or 2 apple-grain aphids did.

These studies showed that, though both aphid species were vectors, the virus (or viruses) transmitted by each species was not the same as shown by severity of disease development.

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RESEARCH STATUS OF SWEETPOTATO INTERNAL CORK VIROSIS IN 1956

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Abstract

The sweetpotato internal cork virosis was discovered in 1944 in South Carolina and has since spread to all the sweetpotato growing States and Territories of the United States. A review of the literature reveals the story on the symptom development, varietal susceptibility, virus nature, graft transmission, cork development in storage, and control measures.

Studies begun at the Plant Industry Station in October 1954 have produced valuable information on the nature and transmission of the causal virus. The incubation period of the virus was reduced from about one year to approximately one week through the discovery of an excellent indicator plant or indexing host, Scarlett O'Hara morning glory. By use of a modification of the Yarwood technique, the virus has been transmitted mechanically. Moreover, the use of a reducing chemical, cysteine, has extended virus activity at room temperature outside the living cell from 0.6 seconds to 2 hours. With the aid of cysteine it is possible to employ mechanical transmission in studies on virus properties designed to develop methods of control. Between mid-September and the following March the indicator plants for cork virus transmission were kept in the proper growth status by photoperiod control in the greenhouse.

Aphid transmission of the internal cork virus was demonstrated from sweetpotato to sweetpotato and from sweetpotato and several morning glory varieties to Scarlett O'Hara, the ideal indexing host.

INTRODUCTION

Internal cork, a new disease of sweetpotato (Ipomoea batatas) of unidentified cause, was first recognized in 1944 in South Carolina by Nusbaum (25). In 1950 he (27) reported the disease to be caused by a virus, having obtained transmission of the necrotic root symptoms by root-plug grafting, after an incubation period of approximately 1 year. By 1950 the disease was known in Maryland, Virginia, North Carolina, South Carolina, Georgia, Alabama, Mississippi, Louisiana, Tennessee and Texas. Subsequently, this disease has been found on the Porto Rico variety and its near relatives throughout most of the other States and Territories growing the crop (Oklahoma (31), California (6), Hawaii (17).

The research status of the sweetpotato internal cork virosis in the United States in 1956 can best be approached by a review of the literature in relation to the life history of the causal agent.

SYMPTOMATOLOGY

Foliage: Nusbaum (26) noted a constant association of a definite succession of foliage symptoms, indicating a virus infection. The foliage had vein feathering and mottling, reddish to purple splotching, sometimes in ring form, and then a gradual fading out of leaf patterns.

Martin (19) found only 1 of 10 lots of sweetpotatoes which originated in 1947 from plants with ringspotting of leaves that produced crops of roots with internal cork symptoms. However, lots originating from internal-cork-symptomed roots consistently produced roots with internal cork. Ringspotting was equally prevalent in all Martin's plots during 3 growing seasons. He suggested the possibility of two causative agents.

It appears that use of the term "ringspot" in the literature has resulted in some confusion. Rankin (28) noted that chlorotic spotting, a more specific term, and vein banding appear as primary symptoms, and anthocyanin pigmentation as a secondary symptom associated with cooler temperatures.

Williams (33) observed four types of foliage symptom development associated with internal cork of sweetpotato: (i) Chlorotic spots, (ii) Chlorotic vein banding, (iii) Chlorotic spots bordered by anthocyanin pigments; and (iv) Chlorotic vein bands with anthocyanin pigments around them. His observations indicate that chlorotic areas may enlarge after initiation and remain until masked by general chlorosis which is associated with senescence. The development of anthocyanin pigment was correlated with low temperatures and the pigment disappeared with an increase in temperature Also, there was higher pigment correlation with temperatures in

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in Porto Rico than in other varieties. Williams noted that pigment is only occasionally formed in the absence of chlorosis.

Roots: Corky areas were observed by Nusbaum (26) to appear in the root flesh about the time the first-formed roots reach marketable size. These areas increase in number and size in the field as the root development progresses. After harvest the cork lesions develop even to a greater extent in storage at favorable high temperatures.

Rankin (28) found a positive relation between sweetpotato root size and the development of internal cork symptoms. Of roots weighing less than 100 grams 40 percent were symptomed or diseased; between 101 and 200 grams, 57 percent were diseased; between 201 and 300 grams, 67 percent were diseased; and of those over 300 grams, 82 percent were diseased. At 55° to 60°F there was practically no symptom development; at 65° very little symptom development; at 70° to 75°, extensive symptom development; at 80° to 85° extensive symptom development plus shriveling.

Williams and Neilsen (34) raised a question concerning the appelation "internal cork" for root symptoms of the disease. They found the freshly cut "internal cork" spots relatively free from cork cambium or true cork. The cork cambium or phellogen produces "phellem," which delimits regions of injury and develops around all wounds. They concluded that small lesions having a well-defined phellem in roots stored at 70° to 80°F are probably caused by some factor other than internal cork virus.

VARIETAL SUSCEPTIBILITY

Relative varietal susceptibility has been difficult to ascertain accurately because symptoms often lack clear-cut definition. Aycock and Hughes (1) observed no internal cork symptoms in roots of two clones, 3-6 and 32-149, which exhibited pronounced ringspotting of leaves. The internal cork virus was, however, transmitted from these clones to cork-free Porto Rico plants by means of core-grafts and cleft-grafts.

Hughes and Aycock (13) found Goldrush susceptible and Allgold resistant to internal cork, although the latter may carry the causal virus without developing root symptoms.

Feazell (4) tested the reaction of sweetpotato varieties and seedlings to internal cork diseases. Mother varieties were core-grafted, sprouts were grown, the crop was harvested and the roots were stored at 70° to 80°F for 7 months and sliced for examination. The varieties rated "Susceptible" were Porto Rico Unit 1, Queen Mary, and Goldrush; "Intermediate", Heartogold; "Resistant", Allgold and Pelican Processor. Louisiana seedlings were also rated: "Susceptible", 9-47; "Intermediate", L-240, 9-2, 9-39, 9-49; "Resistant", 9-18, 9-65, 9-65A, and L-224. When cores were taken from cork-free roots of Heartogold and Allgold varieties and grafted into cork-free Porto Rico roots, internal cork developed markedly in Porto Rico roots. This finding indicates that roots of Allgold and Heartogold may carry the causal virus without exhibiting cork lesions even after 7 months' storage.

Feazell and Martin (5) noted that Earlyport (L-240) was less susceptible to internal cork virus symptoms than Porto Rico. Following core grafting of cork-free mother roots with "corky" tissue, the percentages of roots that showed cork in the resultant crops were as follows: Porto Rico, at harvest 5.9 percent, after storage 89.2 percent; Earlyport at harvest none, after storage 19.6 percent.

Martin (21) compared internal cork development in two varieties grown side by side: Unit 1 Porto Rico and Earlyport. Unit 1 Porto Rico had 25.5 percent cork at harvest and 87.8 percent after storage compared to Earlyport with 0.4 percent at harvest and 45.5 percent after storage.

NATURAL TRANSMISSION

The fact of transmission or spread of sweetpotato internal cork virosis is well known and insect vectors have been assumed to be involved ever since its virus nature was established.

Rankin (28) noted in field tests that plants grown from roots with internal cork symptoms produced varying percentages of corky potatoes. None of the plants had all the roots corky and 16 percent had no roots with internal cork symptoms. Also, expression of symptoms was influenced by environment. In 1949 40 percent of the roots of the Porto Rico variety showed marked symptoms. Seventy-two other varieties and selections were grown for 1 to 8 years exposed to natural transmission and 25 percent produced roots that showed symptoms.

Rankin (29) tested 164 sweetpotato varieties and selections and studied disease transmission by grafting and aphids. In 1950 all but 22 of these selections were eliminated. Tested again in

1951, most of the 22 showed resistance and a few had no internal cork symptoms. In chemical studies no reagent gave a positive reaction to virus presence in host tissue. Soil pH seemed not to influence symptom expression.

Kushman and Deonier (16) surveyed for the incidence of internal cork of sweetpotatoes in Mississippi in 1953 and found it in all sections of the State. Internal cork was present in more than 80 percent of Porto Rico stocks (30 samples), being high in about 1/4 of them. Two samples each of Goldrush and Allgold were collected; the former was affected with internal cork and the latter free of visible root symptoms.

Nielsen (23) reported that internal cork was generally distributed in the Piedmont and Coastal Plain counties of North Carolina by 1947 and 4 percent of all roots examined had corky lesions. In the 1948 crop 12.9 percent of the roots showed disease; in 1952, 71.2 to 90.0 percent. The disease increase was most rapid in the southern and southeastern counties. For study of natural spread, plants from severely infected and also from relatively disease-free roots (0.7 percent) were grown in contiguous plots at three locations for 2 years. At the end of 2 years both lots were equally infected with root lesions.

Transmission by Insects: Rankin's experiments in Georgia indicated that leafhoppers are probably not vectors of sweetpotato internal cork virosis but that aphids (Myzus persicae and perhaps other species) are vectors. Use of steamed plots in combination with unsteamed plots and walk-in cages showed that soil-inhabiting insects are not vectors. Use of several insecticides did not satisfactorily prevent the natural transmission of internal cork virus.

Rankin (29) reported that the internal cork virosis was apparently transmitted by aphids and that octamethylpyrophosphoramide shows promise for control of insect vectors.

Nielsen (23) observed that for an area of high disease potential the pattern of spread into healthy stock is similar to that for viruses with leafhopper vectors and also that sweetpotatoes free of internal cork virus and grown on an isolated coastal island for 2 seasons remained healthy.

Nielsen and Person (24) stated that internal cork increases or spreads into healthy plants or plantings in the Coastal Plain and Piedmont sections of North Carolina if inoculum is nearby. The rate of increase and spread suggests that an insect vector or vectors are present in all sweetpotato growing areas.

STORAGE INCUBATION TEMPERATURES

The endpoint of virus activity is necrosis which is conditioned by growth status. Although a tiny dark brown or black characteristic lesion in a root is a positive symptom of internal cork virosis, the necrotic tissue rarely involves as much as one-half a root after 8 months' storage at high temperatures. At harvest time the incidence of lesions in diseased roots is usually less than 10 percent and is rarely as high as 20 percent. Yet after prolonged storage at warm temperatures the percentage may reach as high as 100 in highly susceptible sweetpotato varieties such as Porto Rico. The reason for this behavior is undoubtedly the nature of the virus itself. Storage temperatures thus play an important role in the incidence of necrotic disease symptoms.

Martin (18) had three lots of diseased sweetpotato roots, with 19.4 percent infection of internal cork at harvest time, that increased to 80 percent after 5 months' storage at favorable high storage temperatures.

Nusbaum (27) devised a method of classifying the measurements and calculating the "cork index" based on different amounts of necrotic root tissue upon removal from storage.

Wilcox and Ezell (32) recorded the development of internal cork in sweetpotatoes at different storage temperatures. At harvest the roots had no cork lesions present. By early June or after about 7 1/2 months' storage at 70°F, 64.8 percent of the roots were corky as compared with 7.4 percent at 60°, and 7.4 percent at 55°.

Nielsen (22) studied the effect of temperature on the development of internal cork lesions in sweetpotato roots. Additional roots develop internal cork lesions and lesions become larger at storage temperatures of 70°, 85°, and 94° F. In contrast no additional lesions develop at 55° Lesions present may enlarge slowly over a 7-month period at 55°. Nielsen calculated a "cork index" based on five size classes from the following formula: Cork index equals

$$\frac{(1n) + (2n) + (3n) + (4n) + (5n)}{N}$$

with "n" being the number of roots in each size class and "N" representing the total number of

roots in a given sample. The cork lesions in cross-section are divided into five size classes:

1 -- < 1/8 inch
2 -- 1/8 inch to 1/4 inch
3 -- 1/4 inch to 1/2 inch
4 -- 1/2 inch to 3/4 inch
5 -- > 3/4 inch

Martin (20) noted a rapid increase in incidence and severity of internal cork lesions during storage at 70° to 80°F, little or no increase at 50° to 60° and a definite increase at 65°. When the roots were stored at 50° to 60° for 150 to 200 days and then put at 80° there was little or no increase in cork lesions during 14 days at 80°. During 36 days at 80° there was some increase in cork lesions but internal cork incidence was low as compared with that for roots held at 80° since harvest. During 58 days after removal from cold storage the cork lesions had increased to a level near that of roots stored continuously at 80°. Martin (21) calculated a "disease index" based on five classes similar to that of Nusbaum (27) and of Nielsen (22).

CONTROL

Usually no control method is feasible until the weak links in the life history of the causal agent are discernible. Consequently, since little is known about the internal cork virosis, very little in the way of control measures has yet been accomplished.

Martin (19) states that his heat treatments of mother roots and vine cuttings were not successful in destroying the agent or agents responsible for the disease symptoms but gives no details.

Rankin (28) noted that several insecticides did not control the transmission of internal cork satisfactorily. Rankin (29) in a progress report observed that antibiotics and chemicals were not chemotherapeutic for internal cork virosis but that octamethylpyrophosphoramide showed promise for insect-vector control.

Holmes (12) reported the elimination of cork virus from sweetpotato foliage by grafting small tip cuttings onto Giant White moonflower (Calonyction aculeatum) and Heavenly Blue morning glory (Ipomoea tricolor). Scions grown to a length of 6 to 18 inches which were free from foliar abnormalities were used to produce a series of larger cuttings. These larger cuttings were then rooted and established as independent subclones.

Johnstone (15) started with nearly if not totally cork-free stock. Roots were bedded and after sprouts were produced the roots were sliced and discarded if cork was present. Plants from cork-free roots were grown in the field and sprayed for aphid control and isolated as much as possible. Harvest was by individual hills.

SYMPTOM, TRANSMISSION, AND INDEXING STUDIES AT BELTSVILLE, MARYLAND

The literature reviewed shows that progress has been slow in uncovering the basic facts about sweetpotato internal cork virosis. Root lesions were the only clear-cut disease symptom recognized, the known incubation period was 1 year and the only transmission method was grafting when the following studies were begun.

Symptom Studies

In early October 1954 in a preliminary study, experimental field plots at the Plant Industry Station were mapped to record diseased sweetpotato foliage symptoms, illustrated in Figure 1. In general, the so-called ring-spot lesions consisted of two kinds: (i) Typical chlorotic spots surrounded by a purplish ring on relatively light green or chlorotic foliage, and (ii) Purple ring-spots usually smaller in size without the chlorotic spot at center on otherwise medium-green leaves. The chlorotic spots appeared first and the anthocyanin rings developed later, toward the end of the season upon the onset of cooler temperatures as the foliage ceased growing. Two factors appeared to be involved, namely lower temperature and maturity. The purple rings alone (minus chlorosis) were not considered to be a result of virus infection but probably of a nutritional condition.

Simultaneously some potted diseased plants growing in the greenhouse were showing the same disease symptoms. The confusing aspect was two-fold: the rather sudden onset of anthocyanin configurations and the almost as sudden disappearance of the clear-cut sharp lesions,

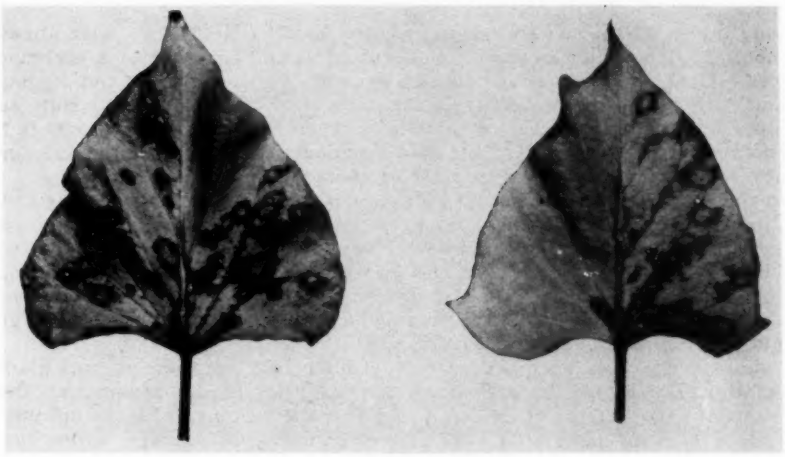

FIGURE 1. Foliage symptoms of internal cork virosis on Porto Rico sweetpotato, Beltsville, Maryland, October 1954

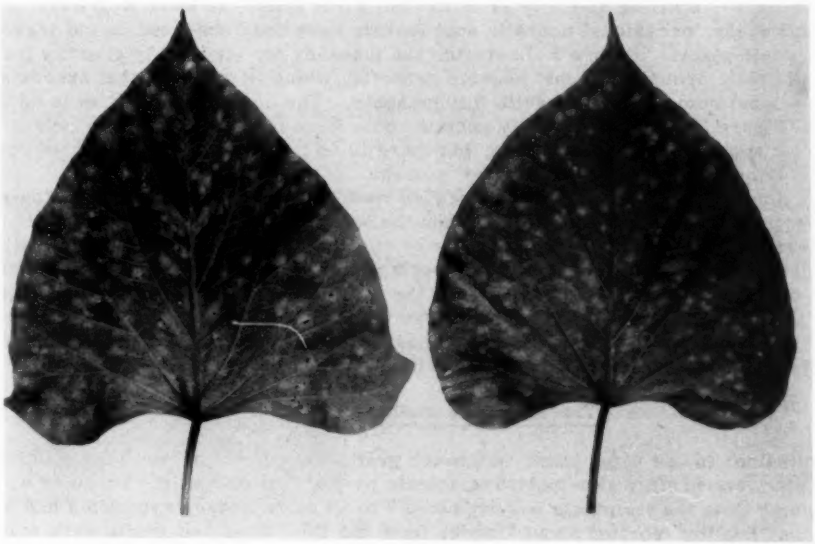

FIGURE 2. Chlorotic leaf spot, a foliage symptom of internal cork virosis on rapidly growing young plants of Porto Rico sweetpotato in the greenhouse. Note that some of the spots are becoming necrotic.

and the tendency for relatively few and usually no more than one or two leaves per terminal, about seven nodes back from the tip, to be so endowed.

After harvest on October 8, the roots were stored for 6 months at 75°F and then sliced for observing cork symptoms. It was then possible to ascertain which root symptoms accompanied what were considered typical, positive foliage disease symptoms of internal cork at digging time the previous autumn. In general, plants having occasional leaves with positive chlorotic spot symptoms also showed positive necrotic spot symptoms on most but not all of the roots in individual hills. This definite correlation of leaf and root symptoms constituted new knowledge of the disease but it developed too slowly to be very useful as a tool.

The inability to check or diagnose symptomed leaves and roots directly showed the great need for a positive indicator plant or indexing host. For internal cork virosis this need was urgent because in sweetpotatoes foliage symptoms were very mild and had a tendency to be masked on most of the leaves. The first important step was to determine symptom development in sweetpotatoes from the beginning to the end of the growing season. A study was designed for the production of a sequence of visible foliage symptoms on the Porto Rico variety in the greenhouse and in the field, in relation to seasonal development.

To start with, some corky roots were bedded in November 1954 and their sprouts planted in rows in a bench of sterilized soil in the greenhouse for observing foliage symptoms. One side of the bench received sprouts from roots containing necrotic lesions, whereas sprouts from symptomless roots from the same hills were planted on the opposite side. At the end of about 1 month, chlorotic spot lesions (1 to 2 mm. in diameter) began to appear on certain leaves, both from the symptomed and the symptomless roots. This was good evidence that whatever induced the chlorotic spots was present in all the roots saved from diseased hills. The absence of such symptoms on growth from known disease-free roots was strong supporting evidence. The foliage was at first normal green and the chlorotic lesions appeared prominent, but shortly there was some fading with loss of contrast as the foliage became lighter in color.

In vigorous sprout growth as many as five or more leaves developed the chlorotic spot symptoms in succession, starting as early as on the third leaf above the base of growth. From the beginning of this study, occasional necrotic spot lesions have been observed on old leaves of diseased sweetpotato plants. Figure 2 illustrates the tendency for vigorously growing leaves to develop some chlorotic spot lesions that become necrotic, which is evidence that necrosis is the end point for at least some of the chlorotic foliage spots. The only root symptom is necrotic or black spots (Figure 3). Ordinarily only certain roots from diseased hills will have reached the necrotic spot stage at digging time, but there is no certainty that all affected roots will show necrotic symptoms even after 8 months' storage.

Cuttings from diseased potted plants were likewise rooted and grown for observing the sequence of foliage symptoms in the greenhouse. When the temperature was kept about 70°F chlorotic spots were followed by purple rings on one or two leaves midway on terminals with about 12 to 15 nodes (Figure 4). In the field in the spring similar chlorotic spots were recorded as present on the diseased cork materials but minus the anthocyanin pigment induced by cool temperatures. Pigment around the chlorotic spots was usually lacking at warm summer temperatures. The close association of the chlorotic foliage spot symptom with corky roots served as the basis for the graft-transmission studies described in the following section.

Transmission Studies

Graft Transmission: In one experiment, approach grafts were made between symptomed diseased and symptomless healthy sweetpotato terminals having 7 to 10 nodes. Three or 4 weeks later, by which time the terminals had produced 7 to 15 more nodes, symptoms had begun to appear on the "healthy" portion about 7 nodes from the tip. When the grafts were made the diseased terminal was showing chlorotic spots with a faint purple ring and 3 to 4 weeks later the same syndrome was reproduced across a graft union as a result of virus transmission. The diseased portion had grown only about one-half as much in length as the healthy portion. However, following the initial appearance of symptoms the foliage color of the recently invaded terminal soon became lighter green. This experiment was run in 8 replicates, of which 6 showed distinctly positive symptoms of chlorotic spot surrounded by purple ringspot, and 2 showed chlorosis without purple ringspot. Thus it was demonstrated that typical foliage symptoms were transmitted by approach grafting within 1 month.

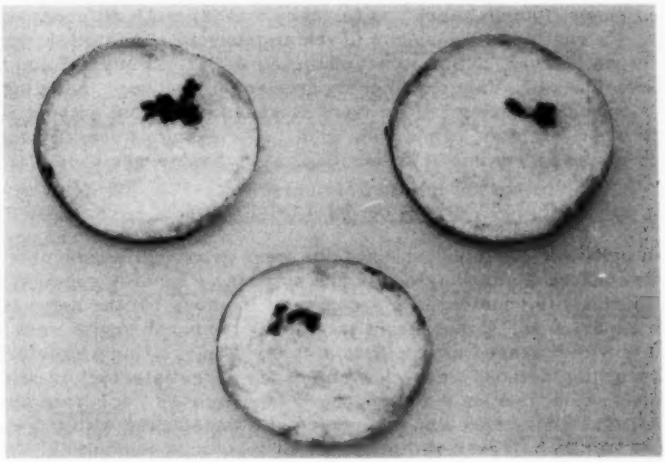

FIGURE 3. Transverse sections of sweetpotato roots showing typical black spot lesions of internal cork disease.

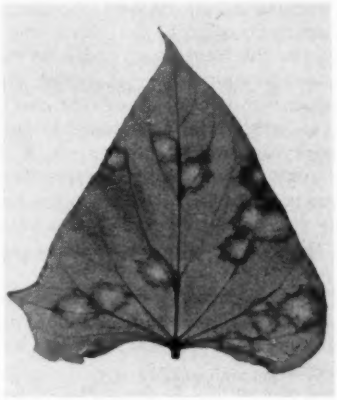

FIGURE 4. Typical Porto Rico chlorotic spot foliage lesions of internal cork being surrounded by purple rings when the greenhouse temperature dropped to about 70°F. or below.

Mechanical-Transmission: Ordinary mechanical methods were first tested for transmission of the sweetpotato viruses (internal cork and feathery mottle) to 10 different species including Nicotiana glutinosa, N. rustica, N. tabacum (Turkish tobacco), tomato (Marglobe), pepper (California Wonder), cowpea (Black), datura, cucumber (A. & C.), Crimson Rambler morning glory, and Porto Rico sweetpotato. Various other transmission methods, including dodder, insect, and "specialized mechanical" using foliage from diseased plants but without requiring the presence of the chlorotic spot symptom, were also tried. The results were all negative except when a specialized rapid modified Yarwood (35) mechanical technique was used (7).

Comprehensive Indexing Experiment

In the search for an indicator plant, about 3 months were required to assemble for testing the various sweetpotato relatives (Ipomoea species and hybrids, Pharbitis species, Quamoclit species, and others. Pharbitis is considered a synonym of Ipomoea but the name is retained for the purposes of this experiment.) In the meantime tip and approach grafts were made on the more advanced test plants with diseased sweetpotato scions. Tip grafting was inferior to approach grafting and all grafting methods were slow and not at all satisfactory in practice.

In February 1955 mechanical transmission of the virus to a series of Ipomoea species and hybrids, Pharbitis species, Quamoclit species, and others was undertaken with infective juice from necrotic root tissue and from leaves with typical ringspots surrounding chlorotic spots, prepared by three methods. The methods were the so-called (i) mortar and pestle, (ii) pot-label, and (iii) modified Yarwood. Earlier failures of mechanical transmission suggested that the virus had been very quickly inactivated outside the host tissue. By practice trials, the exposure of the virus during preparation and transfer was reduced for the three methods to: (i) mortar and pestle, 10 to 15 seconds; (ii) pot-label, 3 to 5 seconds; and (iii) modified Yarwood, 0.6 second. At this short interval the third method was effective with juice from symptomed leaves and roots; the other two methods were ineffective.

By this means Scarlett O'Hara morning glory, an Ipomoea hybrid, was revealed as an ideal indexing host for the virus associated with leaf and root symptoms of internal cork of the sweetpotato (7). By approach grafting the virus was further transmitted in about 3 1/2 weeks from diseased to healthy Scarlett O'Hara. The veinbanding chlorotic mottle symptoms on this plant became evident in about a week after mechanical transmission, in contrast to a month for a vine (approach or tip) graft transmission or a year for root-plug transmission on the sweetpotato.

Subsequent studies have demonstrated that the mechanical transmission of the virus was aided by the use of cysteine, that is, the period of exposure outside the host plant could be increased from 0.6 second to about 2 hours if the tissue to be squeezed was first moistened with 0.1 M. cysteine. Therefore, by this means the regular mechanical or rubbing methods can be used and some of the virus properties determined. Hildebrand (9) also demonstrated that translucent chlorotic spots on Triumph nonpigmented sweetpotato are symptoms of internal cork.

In the indicator plants there was an abrupt transition from vegetative to reproductive development in mid-September and the latter continued until the following March. The indexing host value depends on the continued production of vigorously growing shoots, which could be achieved by subjecting the plants to 3 hours of light during the middle of the night beginning at emergence (10).

Aphid transmission of the causal agent of internal cork virosis has been demonstrated recently by Hildebrand and Smith (11). In Scarlett O'Hara morning glory, an excellent indexing host, the incubation period was 6 to 10 days following transmission either mechanically or by aphids, from sweetpotato, Pharbitis diversifolia, or Scarlett O'Hara.

By the end of the 1955 season indexing of material suspected of containing the virus of sweetpotato internal cork could be accomplished in 10 days or less. More rapid progress toward the understanding and control of the internal cork virosis should now be possible.

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MECHANICAL TRANSMISSION OF PEACH YELLOW BUD MOSAIC VIRUS

C. E. Yarwood

An infection from peaches carrying yellow bud mosaic (1) has been transmitted to cowpea, tobacco, bean, sunflower, cucumber, and guar, (3, and unpublished data). Cowpea is perhaps the most useful of these hosts, since it gives local lesions on the inoculated leaves, a systemic shock infection with necrosis, followed in some cases by recovery with an abundance of virus in the new growth. The infection can be transmitted readily from cowpea to the other herbaceous plants listed and gives the same symptoms as inoculations direct from peaches. Attempts by standard mechanical inoculations and by dodder to transmit the infection from cowpea to peach have been heretofore unsuccessful.

Among the special methods tested was heat treatment of the peach plants before inoculation. This was suggested by the sevenfold increase in infection of beans with tobacco mosaic virus when the bean leaves were heated for 60 seconds in water at 45° C before inoculation (2). The tops of 8 rapidly growing peach trees up to 8 inches in 4-inch pots in the greenhouse were dipped in water at 45° or 50° C for various intervals, removed, dusted with carborundum, and rubbed with juice from infected cowpea. On January 13, 1956, 20 days after inoculation, one peach tree which had been treated at 45° C for 150 seconds before inoculation showed typical early symptoms of peach yellow bud mosaic. Juice from this tree was used to inoculate cowpea and cucumber, and it induced the same symptoms as inoculum from infected cowpea. By February 6, four additional peach trees (one treated at 45° C for 60 seconds, 2 treated at 45° for 90 seconds, and one treated at 50° for 60 seconds) had shown symptoms of peach yellow bud mosaic. In all, 5 out of 8 trees which were heat-treated and inoculated became infected. None of 8 non-heated and non-inoculated trees became infected. Natural (accidental) infection with this virus has never been observed in 2 years of study by the writer, nor in about 15 years of study and observation in the greenhouse in Berkeley by H. E. Thomas.

As only one tree without heat treatment was inoculated in the above series, it can not be safely concluded that the heating of the trees before inoculation contributed to the success of the inoculation of the heated trees, though this is certainly suspected. However, that peach yellow bud mosaic virus can multiply in cowpea, and that this virus can be transmitted to peach by juice inoculation, seem established.

This is believed to be the first case of juice transmission of a virus infection to fruit trees.

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FOLIAGE BURN OF VINIFERA GRAPES AS A SYMPTOM OF WHITE EMPEROR DISEASE

F. N. Harmon and J. H. Weinberger

A disorder of the Emperor variety of grapes that causes the fruit to be light in color and low in sugar and the leaves to be off-color and burned in the early autumn has been observed for many years. Harmon and Snyder (3) proved by graft-transmission studies that this so-called white-Emperor condition is caused by a virus. Hewitt (4) showed that the virus can be transmitted through the wood of four rootstocks, namely 1202, 1613, St. George, and Salt Creek. Subsequently Harmon (2) demonstrated that the white-Emperor virus produces the same fruit and leaf symptoms in the varieties Cardinal and Red Malaga (Maraville de Malaga) as in the Emperor variety. Diseased plants of these three red-fruited varieties have rugose foliage which turns prematurely reddish-brown in the early fall, giving the vines a scorched appearance. Since the white-Emperor virus makes leaves on infected Cardinal and Red Malaga grapes look scorched, infection of vines of other varieties having burned foliage symptoms was suspected.

In the fall of 1954 a survey of the grape variety collection at the U. S. Horticultural Station, Fresno, California, revealed a number of vines on which autumn foliage changes occurred early. Leaf necrosis followed, giving the vines a burned appearance. Thirty-three affected vines in the Fresno collection and 2 similar vines in commercial vineyards, representing 32 varieties and two unnamed seedlings, were selected for indexing. Five buds from each of these source vines were budded into individual bearing Emperor vines in March 1955. In addition, vines of 7 of the 32 varieties which had normal foliage in the fall of 1954 were indexed on Emperor. Alternate unbudded Emperor vines served as controls for the budded vines.

A striking difference in fruit color between Emperor vines budded from burned-foliage vines and unbudded Emperor vines was evident in the fall of 1955, 7 months after budding was done. Ratings on fruit color were made on November 3 (Table 1). The 35 inoculated Emperor test vines averaged 2.86 in fruit color, and no vine had more than a 6 rating. The control vines averaged 8.29, and only one vine, bearing a heavy crop, had less than a 7 rating. The standard error of the difference of 5.43 was .28, highly significant. On the basis of fruit color it is evident that transmission of white-Emperor virus was obtained from all the tested source vines showing burned foliage.

On October 27 and November 4 fruit samples were harvested from each Emperor vine for sugar and acid analyses. White-Emperor disease, in addition to delaying coloring, causes a reduced sugar content of fruit of the Emperor variety. Fruit from the 35 bud-inoculated Emperor vines averaged 12.25 percent sugar content, whereas fruit from alternate control vines averaged 18.35 percent (Table 1). The difference of 6.1 percent had a standard error of .35, highly significant. Transmission of white-Emperor virus from vines with burned foliage was thus demonstrated on the basis of low sugar content of fruit of bud-inoculated Emperor test vines.

Acidity was not appreciably affected by the disease, for samples from budded and control vines averaged .46 and .43 gm per 100 ml of juice, respectively, which is characteristic for Emperor fruit.

Each bud-inoculated Emperor vine had fruit with less color and lower sugar content than its adjacent unbudded vine (Table 1). Since evidence of transmission on a group basis was highly significant statistically, transmission from each source vine was strongly indicated. In all probability each of the 35 source vines with burned foliage was infected with the white-Emperor virus.

Although there was rapid transmission of the white-Emperor virus to the index vines budded from burned-foliage suspect vines, no burned-foliage symptoms developed on the index Emperor vines during the first year. It appears that fruit was more quickly affected than foliage by the virus.

Seven vines which had healthy foliage were indexed on Emperor but only six of the test vines bore fruit; thus comparisons can be made for only six cases (Table 2). The data for vines with burned foliage are repeated from Table 1, for comparisons with transmission from vines with healthy foliage. Inoculations from healthy-appearing Black Hamburg, Calmeria, Red Malaga, and Thompson Seedless (Sultanina) vines failed to reduce color and sugar content of fruit on indicator vines appreciably below those of fruit on unbudded controls. Apparently no transmission was effected. Buds from the unburned G-4-74 vine caused reduced fruit color and sugar content of the Emperor test vines to nearly the same degree as buds from the

Table 1. Fruit color and sugar and acid contents of Emperor grape vines budded from burned-foliage vines and of adjacent unbudded (control) vines.

Source variety or seedling	Fruit color[a]		Sugar content (percent)		Acid content gm per 100 ml of juice	
	Budded vines	Unbudded vines	Budded vines	Unbudded vines	Budded vines	Unbudded vines
Agadia	5	10	16.7	19.4	.39	.44
Ah Saibe	4	9	12.2	18.5	.52	.51
Black Hamburg	3	10	12.1	20.9	.39	.45
Black Seedless	1	9	10.1	21.4	.61	.49
Boal de Madeira	2	9	13.7	18.6	.49	.45
Burger	5	9	11.5	20.6	.46	.57
Calmeria	6	8	16.3	18.3	.46	.41
Chali Sar	2	8	11.2	18.7	.36	.47
Corinthe Blanc	2	4	11.9	16.1	.51	.49
Corinthe Rose	1	8	9.3	18.1	.57	.48
Diamond Jubilee	1	9	10.9	18.5	.51	.51
Emperor	4	8	13.3	18.1	.34	.31
Flame Tokay	2	8	12.2	17.5	.43	.42
Flame Tokay x Ribier	3	7	13.3	16.9	.51	.46
Foster	2	7	12.2	17.8	.51	.49
G-4-74	2	8	11.8	18.8	.42	.37
Keropodia	2	8	9.8	18.8	.47	.51
Koshu	2	8	11.8	19.2	.52	.46
Malvasia de Brogliana	4	8	13.6	18.1	.46	.46
Mamelon	3	7	13.1	17.4	.44	.44
Monake	2	10	10.0	15.8	.29	.28
Mondeuse	3	8	14.3	19.5	.46	.50
Palomino	3	8	12.6	16.4	.36	.40
Pinot Noir	3	9	11.7	17.2	.42	.37
Prune de Cazouls	5	8	12.3	17.7	.47	.42
Red Malaga	3	10	13.1	20.9	.42	.45
Red Malaga	2	9	11.7	17.7	.50	.42
Roussane	4	7	12.9	17.5	.46	.47
Sauvignon Vert	4	8	12.6	20.2	.53	.52
Semillon	3	10	10.7	20.3	.47	.48
Serektia	2	7	9.5	15.9	.48	.44
Servan Blanc	3	8	12.4	17.4	.46	.46
Thompson Seedless	2	7	13.4	20.6	.42	.57
Tinta Amerélla	2	9	12.9	17.7	.47	.41
White Malaga	3	10	11.8	15.8	.40	.28
Average	2.86	8.29	12.25	18.35	.46	.43

[a] 1 = No color; 10 = most color.

Table 2. Fruit color and sugar content of Emperor grape vines budded from vines with burned and healthy foliage and of adjacent unbudded (control) vines.

Source variety or seedling	Fruit color [a]			Sugar content (percent)		
	Vines budded from burned-foliage source	Vines budded from healthy-foliage source	Vines not budded	Vines budded from burned-foliage source	Vines budded from healthy-foliage source	Vines not budded
Black Hamburg	3	8	10	12.1	17.2	20.9
Calmeria	6	8	8	16.3	20.0	18.3
G-4-74	2	3	8	11.8	14.2	18.8
Red Malaga	3	10	10	13.1	19.0	20.9
Red Malaga	2	6	9	11.7	16.1	17.7
Thompson Seedless	2	8	7	13.4	18.8	20.6

[a] See Table 1.

G-4-74 vine with burned foliage. Apparently the G-4-74 source used was infected and transmitted white-Emperor disease even though its foliage was not burned.

Comparisons between vines proved diseased or healthy by transmission tests were possible in four varieties. Fruit color was affected by the disease in three colored varieties, Emperor, Red Malaga, and Black Hamburg, but not in the white variety Calmeria. Foliage was burned and fruit sugar was reduced in all four varieties by the disease. Differences in fruit maturity on diseased and healthy vines were apparent in the two red-fruited varieties, but there was no difference in external appearances in the Calmeria variety in October observations. When fruit of Black Hamburg appeared mature on the diseased vine it appeared overripe on the healthy vine.

Foliage burn and lower sugar content of fruit were the principal symptoms of the white-Emperor disease in the white and intense varieties, whereas in the red varieties failure to develop normal fruit color was also diagnostic. Along with a lower sugar content was a delay in maturity of the fruit. Some diseased vines of the early-maturing Cardinal attained satisfactory fruit color and maturity in late September, approximately 2 months later than on healthy vines. The late-ripening Emperor variety failed to attain full maturity on diseased vines because of the limits of the growing season.

It has been suggested that foliage burn alone might be responsible for the reduced sugar content and late maturity of fruit. That this is not always the case has been illustrated several times where Emperor fruit on canes originating near the point of inoculation was white and late in maturing, although the fruit on the remainder of the vine was red and normal and none of the foliage was burned. Also in the Cardinal variety late coloring of fruit on the diseased vines was evident before much if any foliage burn occurred. It was evident that low sugar content and late coloring of fruit were not secondary effects, but primary symptoms of the disease. Other viruses are known to affect phloem structure, interfering with translocation (1). White-Emperor virus may have a similar effect in grapes.

Where early autumnal foliage burn occurs on vinifera grapes with no apparent cause of the disorder, such as soil deficiencies, the presence of white-Emperor disease may be suspected. This is particularly true where vines are grown on rootstocks.

Sample surveys made in ten commercial vineyards on rootstocks near Fresno, in the fall of 1955, showed much variation in foliage burn but approximately one-fourth of the vines had severely burned foliage. Two of the cases of transmission of white-Emperor disease reported here were from burned-foliage vines in commercial vineyards. Further indexing of commercial vineyards is in progress. Industry criticism of certain rootstocks may be partially due to white-Emperor virus in the stocks.

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PEACH CANKER (FUSICOCCUM AMYGDALI): TIMES AND SITES OF INFECTIONS

Daniel F. Cohoon and Robert H. Daines[1]

SUMMARY

Nodal cankers on peach trees produced by Fusicoccum amygdali appear throughout the year, with the greatest number occurring in the spring and fall. Inoculation experiments show that twig infections occur at bud scales, stipules, fruit and leaf scars, and at the blossoms. Orchard and greenhouse studies reveal that twig infection does not result from invasion of the twig through the leaf and leaf petiole. Temperature studies indicate that resistance to leaf scar infections develops more rapidly at 80° F than at lower temperatures. These studies also reveal that the leaf scars of the susceptible variety Raritan Rose remain susceptible to infection for a longer period than do those of the less susceptible Sunhigh variety.

Peach canker (Fusicoccum amygdali Delacr.) first reported by Haenseler and Daines (3), has in recent years threatened the future of many peach varieties grown in southern New Jersey. The study reported here was undertaken to learn where and when infections occur as a basis for disease control studies.

DESCRIPTION OF DISEASE

Since a detailed description of the wood canker phase of peach canker has been given by Daines and Cohoon (1) and by Guba (2), only brief mention of these cankers will be given here.

Wood cankers at the buds and the bases of twigs frequently result in girdling and death of the distal portions. In young non-bearing trees such cankers may partially or completely girdle the trunk or structural limbs. When the girdling is complete, death of the tree or limb results; when not complete, a flat-sided weakened area develops.

The symptoms and signs of this disease were first observed on the peach leaves and fruits by the authors during the summer and fall of 1952, and were described by them in 1953 (1). Later, Guba (2) confirmed the occurrence of this disease on peach foliage. Although leaf infections do not seem to result in twig cankers, they do have the effect of greatly increasing the amount of inoculum present during late summer and fall.

EXPERIMENTAL METHODS

In this investigation, inoculation studies were conducted in the orchard and in the greenhouse. Spore suspensions were applied to specific locations with a camel's hair brush, and also to limbs or entire trees as a spray. Moist films were maintained by wrapping the twig in moist cotton covered with tinfoil, or by the use of humidifiers. The spores used were obtained from exuding tendrils on twig cankers. For defoliation studies the leaf blade was removed by cutting; only the leaf petiole was left attached and within 2 days these petioles loosened and fell. This method was chosen in preference to chemical defoliants in order to avoid any possible chemical interference with normal callus formation. The orchard counts on canker development were made from labeled twigs at 2-week intervals. As data were recorded, cankers were nicked with a knife, to make certain that they would not be included in future counts.

RESULTS OF EXPERIMENTS

Infection Sites: Inoculation experiments in the greenhouse showed that if a wound is provided infections occur readily on all varieties. Conversely, inoculated unwounded internodal areas consistently remained healthy. Inoculation experiments showed that infection may occur at fresh leaf scars, bud scale scars, stipule scars, fruit scars, and also through the blossoms. On the leaves and fruits, infection occurs at wounded areas, where worm holes and bacterial spot injury were observed as the sites of fruit infection in nature.

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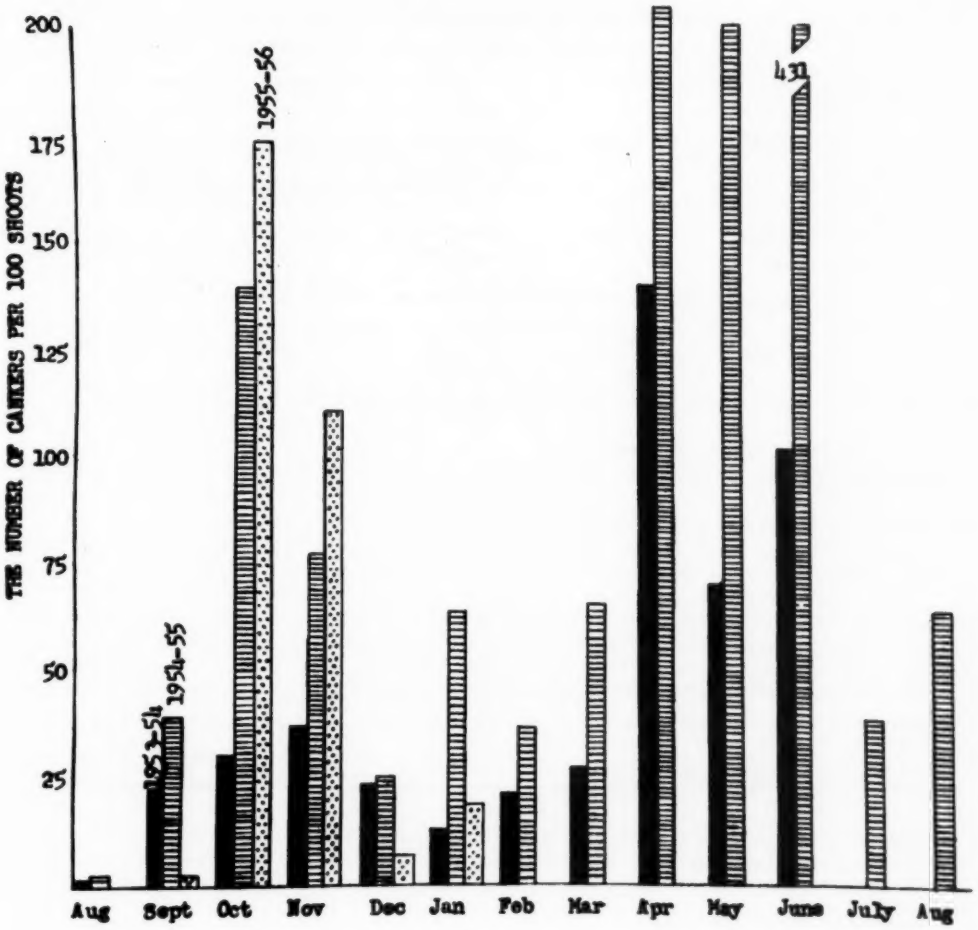

FIGURE 1. The number of new Fusicoccum cankers to develop on peach in an unsprayed Raritan Rose planting. New Jersey, 1953-1956.

Table 1. Effect of time and temperature, after defoliation, on leaf scar infection of two peach varieties by Fusicoccum amygdali.

Variety	Temperature	Days between defoliation and inoculation (a)				
		Immediately	6	12	28	37
			Percent infection			
Raritan Rose	40°F.	23.3	18.3	27.7	11.6	4.9
	80°		5.9	10.3	3.7	3.2
Sunhigh	40°	10.0	7.7	12.0	8.5	0
	80°		4.7	4.6	2.3	0

(a) L.S.D. 5% level 6.0
1% level 8.0

Table 2. Effect of tree development on infection by Fusicoccum amygdali.

Stage of development at Inoculation	Buds Showing Cankers (a)	New Shoots Showing Basal Cankers
	%	%
Dormant	6.8	2.4
Vegetative buds just showing green	8.0	3.5
Vegetative buds 1/2" green	12.0	17.2
Leaves unfurling	22.6	31.0
Vegetative buds 1" to 3" green	25.3	39.6
Vegetative shoots starting to elongate	25.0	43.0
Vegetative shoots 4" to 6" long	47.8	50.0
Vegetative shoots 8" long	27.5	41.0
Vegetative shoots 8" to 10" long	6.7	10.0
Vegetative shoots 8" to 12" long-maturing	1.9	4.4

(a) Includes all types of cankers

Leaf Infections: Leaf lesions tend to be V-shaped with an infected vein or veinlet occupying the apex of the V. Infected leaves develop chlorosis and are shed prematurely. The fungus progresses most rapidly in the veins and can frequently be recovered from vascular elements in advance of the visibly affected areas. To determine whether the fungus enters the twig by way of the leaf, healthy and infected leaves were tagged in the orchard and leaves were inoculated at various locations in the greenhouse. Two groups of diseased leaves were tagged: group 1, terminally infected; and group 2, infected near the petiole. Healthy leaves were also tagged. When tagged nodes were examined for cankers during the following spring, the percentage with cankers was greater for nodes with healthy than for nodes with diseased foliage. This indicated that twig infections seldom, if ever, result from infection of the foliage first and subsequent advance into the twig via the vascular elements. The fact that the nodes with diseased leaves attached developed fewer cankers than those with healthy leaves suggests that early defoliation favored the development of resistance at the leaf scars.

Time of Infection: In an unsprayed, severely infected, Raritan Rose block during the years 1953 to 1955 inclusive, 200 disease-free current season shoots were labeled each fall and examined at 2-week intervals for the appearance of cankers. Figure 1 gives a summary of the results. It is clear that although new cankers develop throughout the year, the maximum number appears during spring and fall.

Spring Infections: To determine the period in the spring when infections are most likely to occur, five potted Raritan Rose peach trees for each stage of development indicated in Table 2 were sprayed with a heavy spore suspension and placed in a moist chamber where the temperature was 80° F and the relative humidity held at 90 percent. After 3 days, the trees were removed and placed on a bench in the greenhouse. Canker counts and types of symptoms that developed were recorded at intervals.

From this experiment, it was learned that infections that occurred during the bud-swelling time either killed the buds before they opened, or after the blossoms had just opened when the leaves were beginning to unfurl, or after the development of very short shoots. Later infections resulted in an increase in the percentage of cankers located at the base of the current season's twig growth. It frequently took a month or more from the time of infection for these cankers to appear. Many such terminals were not killed by the canker for a period of 3 to 4 months. This type of canker corresponds to the basal canker that appears in the orchard in the summer and fall and causes the death of twigs during late summer, fall, and winter months.

Summer Infection: To determine whether infection of 2-year wood may occur through fruit scars, orchard counts and fruit-scar inoculations were made. From counts of labeled fruit-bearing nodes in an unsprayed Raritan Rose orchard, it was found that 6 percent of the fruit scars became infected following the June drop, and nearly the same percentage of scars developed cankers following the harvest period. Inoculation of fresh fruit scars resulted in cankers at 44 to 55 percent of the scars, whereas uninoculated check scars showed infection varying from 8 to 12 percent.

Fall Infection: The canker counts made at 2-week intervals (Figure 1) on labeled twigs revealed that new cankers at the nodes on current-season twigs continue to appear during the fall and winter. Spore inoculations at the nodes made during this period resulted in infections throughout the fall and early winter. The percentage of nodes to become infected as a result of these inoculations decreased as the season progressed, with new infections ceasing in January.

To determine the effect of temperature on the development of resistance to infection, potted peach trees of two varieties, Raritan Rose (very susceptible) and Sunhigh (moderately resistant) were defoliated and then placed in rooms held at temperatures of 80° and 40° F. After various periods of time, trees in both varieties were removed, sprayed with spores, and placed in a room with relative humidity varying from 90 to 95 percent and a temperature of 80°. After 72 hours, the trees were removed, placed in the greenhouse, and examined from time to time for cankers. This study (Table 1) revealed that the leaf scars of each variety became increasingly more resistant to infection as the time interval after defoliation increased. It is of interest to note that the leaf scars of both varieties developed resistance in a shorter period at 80° than at 40°; and that the least susceptible variety, Sunhigh, developed sufficient resistance at both temperatures during the course of the experiment to prevent infection.

A similar experiment was conducted under 54° and 67° F post-defoliation holding tempera-

tures. Since the number of available trees was too small for statistical analysis, data are not presented here. However, it is of interest to know that the trees held at the higher temperature consistently showed fewer infections than the group held at 53°. Our studies on resistance to canker will form the subject of a future paper. The apparent differences in susceptibility of leaf scars to infection at defoliation, and the effect of temperature on susceptibility, may partially explain the differences in disease severity that occur from year to year.

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FUSARIUM STALK ROT OF BANANAS IN CENTRAL AMERICA

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Stalk heart rot of bananas (Musa sapientum) and abaca (M. textilis) in the Asiatic tropics is often associated with the virus diseases bunchy top and mosaic. In the American tropics, stalk heart rot has been ascribed to fungi and bacteria. Several workers in the Philippine Islands have reported consistent isolation of Fusarium moniliforme Sheldon var. subglutinans Wollenweber & Reinking from diseased heart tissue. Ocfemia and Mendiola (1) and Ramos (2) obtained infection with this fungus in leaf inoculations of abaca. Wardlaw (4) described heart rot in bananas in Trinidad and observed F. moniliforme var. subglutinans on the surface of diseased tissue. Later, Reinking (3) isolated F. moniliforme from affected banana plants in Honduras, noting particularly chain production of microconidia in all cultures. His work, however, did not include inoculation experiments with this fungus.

The three hosts most commonly affected by stalk rot in Central America are banana, abaca, and plantain (M. paradisiaca). External symptoms are typified by dying and rotting of the central or inner leaves. These may remain folded or become nearly fully unfurled as they emerge from the crown. Dying leaves are bright yellow or brown in comparison with the normal green of healthy leaves. Occasionally, adjacent green leaves collapse at the petiole base. In advanced cases, all leaves decline and collapse until the entire stalk or pseudostem, which in musaceous plants is formed of leaf sheaths, is killed. Generally only one pseudostem within the mat or clump is affected. However, other adjacent mats may contain diseased plants.

Internal symptoms of pseudostem rot are more variable. In most cases, the developing leaf laminae still furled within the pseudostem heart are blackened and decaying (Fig. 1). This rot, which may be firm and odorless, may extend to within 2 feet of the pseudostem base while surrounding tissue remains healthy and non-affected or only slightly yellowed. With advancing secondary bacterial invasion, the central tissue breaks down and the hollowed pseudostem fills with a foul-smelling liquid. Soft rot rapidly spreads to the upper part of the rhizome.

Healthy sheath surfaces in contact with rotting sheaths within the pseudostem are often streaked brown or purple. Less frequently, this discoloration extends into the parenchymatous ribs of the sheaths. Infected leaves developing in the pseudostem usually are twisted and aborted, forming an obstruction to the follower leaves which are damaged and decay. This tissue is then rapidly invaded by secondary rot organisms.

Occasionally a ring rot forms in the pseudostem with the youngest or central sheaths and the outer sheaths remaining healthy (Fig. 2).

Ring rot infections are often arrested as outer sheaths are sloughed off and a new healthy pseudostem is formed.

A pocket rot may develop as scattered areas of dark purple or nearly black pockets of decay throughout the pseudostem (Fig. 3). These areas remain relatively free of secondary soft rot.

Recovery of central sheaths from heart rot is rare and can only occur with actively growing, young plants when the growing point has not been damaged or when growth is rapid through the decayed area. However, recovery has been observed in plants damaged by floods and windstorms or cut back in pruning operations. These plants present a ragged appearance because the leaf laminae developing within the pseudostem have been attacked by fungi and bacteria. Further lamina rot is stopped when leaves emerge from the crown (Fig. 4).

Fusarium moniliforme sporulates heavily on surfaces of diseased sheaths that have not been attacked by secondary soft rot. Yellow streaks and brown or purple blotches on these surfaces have also yielded pure cultures of this fungus, which has not, however, been isolated from healthy tissue below the diseased area. When soft rot extends into the lower pseudostem, vascular discoloration may appear in the upper rhizome as well as in the pseudostem itself in some instances. F. moniliforme, in association with Cephalosporium spp. and Fusarium solani, has been isolated from this discolored tissue, while ring rot and pocket rot areas have yielded F. moniliforme only.

All isolates produced microconidia in false heads typical of Fusarium moniliforme var. subglutinans. However, microconidia in chains as well as in false heads are infrequently produced in the same colony. Consequently, the fungus may more aptly be identified as F. monili-

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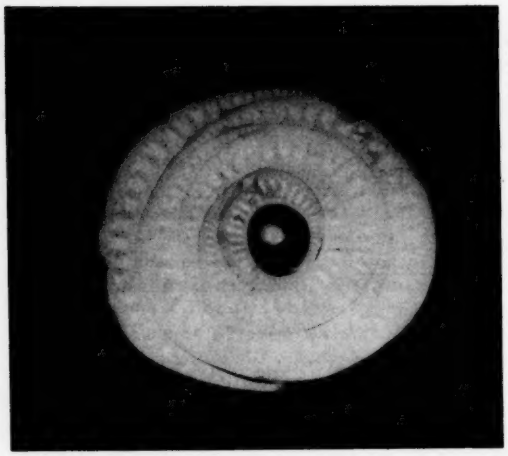

FIGURE 1.
Heart rot in abaca (Musa textilis, var. Maguindanao) confined to center of the pseudostem.

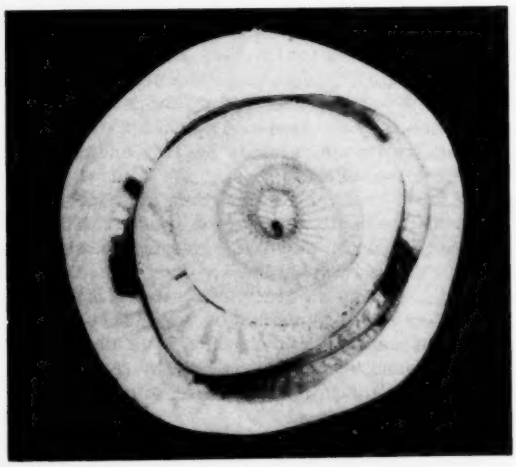

FIGURE 2.
Pseudostem ring rot in banana (Musa sapientum, var. Gros Michel). Inner and outer sheaths remain healthy.

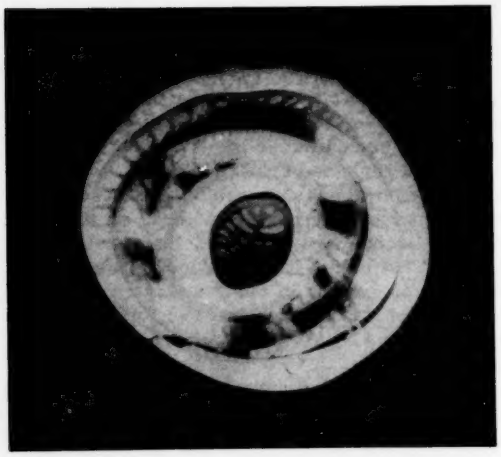

FIGURE 3.
Scattered purple or black pocket rot in plantain pseudostem (Musa paradisiaca, var. Chato).

FIGURE 4.
Recovery of banana from pseudostem rot following flood damage and subsequent pruning. Note ragged leaf lamina attacked by secondary organisms.

Soil inoculation with spores of F. moniliforme did not produce rot of banana, plantain, or abaca plants in boxes, nor did spore suspensions poured into the crowns of these plants between leaf sheaths. Infection in the heart area near the crown resulted only when sheaths or furled leaves in the pseudostem were wounded by a needle, enabling the fungus to penetrate this tissue. Rot spread most readily in furled leaf laminae and progressed into midribs in later stages. Similar inoculations with pure cultures of bacteria associated with F. moniliforme rot have failed to cause infection.

F. moniliforme occurs commonly as a saprophyte on dry leaves, cut pseudostems, or in plant trash in banana farms. The fungus is occasionally found in pseudostems, rhizomes, and roots of plants infected with Pananma disease (Fusarium oxysporum f. cubense) and Moko disease (Pseudomonas solanacearum). Laboratory and field tests have indicated that aerial spores are not easily dislodged from hyphae by wind alone but are readily removed by water. Thus, distribution of spores in the field probably occurs only through wind-driven rain or overhead irrigation.

Stalk rot in banana plantations in Central America is prevalent in young plantings before the first fruit crop is produced, in wind-damaged plants, and in severely pruned mats. For reasons unknown, disease incidence is often high in newly planted areas intentionally flood-fallowed previously for the eradication of the Panama disease Fusarium. However, progressively less disease appears in older plantings in these areas. Removal of diseased stalks is done during regular pruning cycles since the disease seldom becomes a problem in commercial banana plantings.

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1955 BEAN DISEASE SURVEY IN SOUTHERN IDAHO

V. E. Wilson

The present survey of bean diseases was made in the important bean-growing areas of southern Idaho. Bean fields in Canyon, Cassia, Jerome, Minidoka, and Twin Falls Counties were inspected during June, July, August, and early September.

Bacterial Blight (Xanthomonas phaseoli (E. F. Sm.) Dows.): Common bacterial blight occurred in trace amounts in certain fields in the Twin Falls and Delco areas. Bean fields near Hansen and Murtaugh planted with seed of the Michelite variety grown in Michigan in 1954 showed nearly 100 percent infection.

Root Rots (Fusarium solani f. phaseoli (Burk.) Snyder & Hansen and Rhizoctonia sp.): These diseases are prevalent throughout Idaho as well as most other Western States where beans are widely grown. These maladies were less severe in southern Idaho in 1955 than in other years, probably because of warmer temperatures that prevailed during most of the growing season.

Pythium Wilt (Pythium butleri Subr.): This disease was general in southern Idaho. Two fields in the Hazelton area showed about 30 percent infection. Pythium wilt is favored by warm temperature and high humidity, but occurs in arid country where heavy irrigation may provide the necessary moisture condition.

Sclerotinia Wilt (Sclerotinia sclerotiorum (Lib.) DBy.): Sclerotinia wilt was widespread in in southern Idaho. Three fields in the Tuttle area, planted to beans for 4 consecutive years, showed 30 percent infection. High incidence of the disease was observed in the Rupert, Caldwell and Twin Falls areas. Under field conditions, rank growth of bean plants and heavy irrigation are correlated with the development of this disease.

Virus Diseases: Common bean mosaic (bean virus 1) was present in all areas of southern Idaho. Disease expression was late and yields were not markedly reduced.

Yellow bean mosaic (bean virus 2) was not severe in 1955. A small number of diseased plants were found in commercial plantings as well as in experimental plots at the University of Idaho Twin Falls Branch Agricultural Experiment Station.

Curly top was infrequent in occurrence. Low populations of beet leaf-hopper (Circulifer tenellus (Baker)) vectors during 1954 and 1955 reduced the incidence of this disease.

Red node, caused by a strain of tobacco streak virus, appeared in trace amounts in snap and dry bean varieties.

An unidentified bean mosaic, resembling common bean mosaic but occurring in Great Northern 123 and Great Northern 59, was first observed in 1954. Trace amounts of this mosaic appeared in these varieties again in 1955.

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AN OUTBREAK OF TOMATO LATE BLIGHT IN PLASTIC GREENHOUSES

C. W. Roane and P. H. Massey, Jr.[1]

The occurrence of tomato late blight (Phytophthora infestans) in a plastic greenhouse near Blacksburg, Virginia, indicates the need for careful management and modification of the design of these structures. Plastic greenhouses are being used in rapidly increasing numbers in Virginia because their initial cost is low and their operation fulfills a service needed by many small communities which could not finance a house of glass construction. These houses, however, are of much tighter construction than conventional houses and for that reason the prevention of excessive moisture is difficult during cold weather.

The operators of the local plastic houses attempted to grow a crop of tomatoes during the winter of 1955-56 but were thwarted by an outbreak of the late blight disease. Plants were started outside and transplanted to the greenhouse about September 1. The greenhouse was covered with polyethylene plastic on October 1 and approximately 45 days later late blight was observed. Despite efforts to control it with fungicides, the disease continued spreading to stems, flower clusters, and fruits. As the outside temperature dropped, moisture control became more difficult. Low night temperature in December was accompanied by lower operating temperatures, whereupon the houses became an incubator favoring late blight development. It was economically not feasible with the use of bottled gas to maintain night temperatures above 55°F. It was impossible, therefore, to ventilate the houses properly and at the same time maintain suitable temperatures in them. Foreseeing a total loss from late blight, the grower pulled out the plants and stopped the operation for the winter. Late blight did not develop on plants in an adjacent house where the tomatoes were set out after the house was covered with plastic.

From a horticultural standpoint, several improvements in design are necessary before these plastic houses can be used satisfactorily for year-round operation. These changes should be directed toward the control of excessive moisture. The bottled gas heating units now being used in plastic houses do not have the capacity to permit proper ventilation during sub-freezing weather.

From a pathological standpoint, the plastic greenhouse should make an excellent incubation chamber for use in large scale disease-resistance studies. Since they can be built in various sizes and shapes and heated individually, they may also be used advantageously to supplement glasshouse space that is now overcrowded or in which isolation is impossible. In addition, they may be constructed at a low cost by unskilled labor[2].

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[2]Massey, P. H., Jr., and H. H. Gee. 1955. Plastic greenhouses. Virginia Agricultural Extension Service Circular 663.

THE INFLUENCE OF OVERHEAD IRRIGATION ON THE INCIDENCE AND CONTROL OF CERTAIN TOMATO DISEASES[1]

D. F. Crossan and P. J. Lloyd[2]

Summary

The relationship between overhead irrigation and the incidence, severity, and control of certain tomato diseases was investigated in field plot experiments. Plots receiving four irrigations totaling approximately 4 inches of water in a 30-day period had a significantly higher incidence of tomato anthracnose (Colletotrichum phomoides) and fruit rot (caused primarily by Rhizoctonia solani) and a significantly lower incidence of blossom-end rot. The fungicidal materials, maneb and zineb, gave signficant control of anthracnose under both irrigated and non-irrigated conditions, but maneb was a more satisfactory material than zineb under irrigation. Both materials appeared to decrease the severity of blossom-end rot in non-irrigated plots, but neither material gave control of fruit rots due to Rhizoctonia solani.

A relationship between the use of overhead irrigation and the incidence and severity of various diseases on numerous crops has been observed in recent years but has only occasionally been experimentally demonstrated (2, 3). In addition, information is lacking on the influence of irrigation on the effectiveness of the fungicides and methods of application currently recommended for control of given diseases under non-irrigated conditions.

Growers in Delaware who have irrigated tomatoes during the past few years have reported an increase in certain disease problems, particularly anthracnose caused by Colletotrichum phomoides. In 1955, an experiment was conducted to determine whether or not: (i) the incidence and severity of tomato anthracnose was greater under irrigated conditions; (ii) fungicides currently recommended for control of tomato anthracnose were effective under irrigated conditions; (iii) the incidence and severity of other diseases increased or decreased.

MATERIALS AND METHODS

A split-plot design, replicated in triplicate, with irrigation treatments as the main plots and fungicidal treatments as sub-plots was used. The tomato variety used was Garden State. Each plot consisted of three treatment rows of 10 plants each. Two fungicides, maneb[3] (Dithane M-22) and zineb[4] (Dithane Z-78), were compared with an untreated check in each plot. Both trade-named materials were used at a concentration of 2 pounds per 100 gallons of water, and approximately 125 gallons of spray was applied per acre per application. The plots were sprayed weekly, starting July 7 and ending August 15, 1955 for a total of 6 applications. In the case of the irrigated plots, the fungicides were applied one day prior to each irrigation.

Overhead irrigation was accomplished by use of 6-foot "Rainmakers"[5], at 20 pounds water pressure, which irrigated a 30 by 40 foot area. The distribution of water over the plot area was relatively uniform. The plots received four irrigations of approximately 1 inch of water per plot per irrigation on July 5, 12, 19, and 26. The plants were flowering and had initial sets of small fruit prior to the first irrigation and fungicidal application. As 0.75 inch of natural rain occurred between July 1 and August 10, and heavy rains occurred during the period August 11-14, inclusive, no further irrigations were made. Harvest began on August 1 and continued until September 8, 1955.

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[1] Published as Miscellaneous Paper No. 240 with the approval of the Director of the Delaware Agricultural Experiment Station. Contribution No. 74 of the Department of Plant Pathology.

[2] Assistant Research Professor and Graduate Research Assistant, respectively.

[3] maneb (manganous ethylene bis [dithiocarbamate]). Supplied by Rohm & Haas Company, Philadelphia, Pennsylvania.

[4] zineb (zinc ethylene bis [dithiocarbamate]). Supplied by Rohm & Haas Company, Philadelphia, Pennsylvania.

[5] March Irrigation Company, Muskegon, Michigan.

Table 1. Influence of overhead irrigation on incidence and control of tomato anthracnose.

Treatment	Concentration (lbs.-gals.)	Number of infected fruit picked[a] (8/1 to 8/15/55)	
		Non-irrigated	Irrigated
Check	----------	43	124
Zineb	2-100	4	67
Maneb	2-100	2	27
L.S.D. @ 1% P. for treatment: 11			
[a]Total M'ktable fruit (3 reps. each):		1185	787

Table 2. Influence of irrigation and natural rainfall on the incidence of tomato anthracnose.

Harvest date	Number of infected fruit	
	Irrigated	Non-irrigated
Aug. 1	15	1
10	138	5
- - - - - - - - 8.18" rain -		
15	65	43
22	243	123
26	60	36
Total	531	208

Table 3. Influence of irrigation on incidence of blossom-end rot of tomato.

Treatment	Number of diseased fruit[a]	
	Non-irrigated	Irrigated
Check	70	3
Zineb	35	0
Maneb	38	2
L.S.D. @ 5% P. for treatment: 10		
[a]Total marketable fruit picked 8/1-9/8	1982	2254

Table 4. Influence of irrigation on marketable yield of tomatoes.

Treatment	Weight (lbs.) 8/1-9/8/55	
	Non-Irrigated	Irrigated
Check	175	198
Zineb	200	268
Maneb	219	349
Total	595	813
L.S.D. @ 5% P. for treatment: 16		

Table 5. Influence of irrigation on the incidence of Pythium and Rhizoctonia fruit rots (8/1 to 9/8/55)

Treatment	Total number of fruit infected[a]			
	Non-Irrigated		Irrigated	
	Pyth.	Rhiz.	Pyth.	Rhiz.
Check	0	7	5	40
Zineb	0	3	3	46
Maneb	0	5	1	49
Total	0	15	9	135

[a]Total marketable fruit picked 8/1-9/8: Non-Irr. = 1982; Irr. = 2254

EXPERIMENTAL RESULTS

It was apparent at the first harvest of red-ripe fruit that anthracnose was prevalent in the irrigated plots but practically non-existent in non-irrigated plots (Table 1). This relation continued to hold true until the middle of August. By August 22 the incidence of anthracnose increased in the non-irrigated plots following the hurricane rains of August 11-14, inclusive. The ratio between the number of infected fruit from irrigated versus non-irrigated plots decreased from a high of approximately 27:1 to about 2:1 (Table 2).

Initially the yield of ripe fruit was greater from non-irrigated plots due to premature ripening under drouth conditions but at the same time there was a proportionately larger number of green fruit on the irrigated vines. This was reflected in the final total marketable yield.

It is apparent from the data that: (a) irrigation and natural rainfall significantly increased the incidence of anthracnose; (b) the maneb fungicide was significantly better than the zineb fungicide for control of anthracnose under irrigated conditions whereas under non-irrigated conditions both materials gave essentially the same degree of control; (c) both fungicides gave significant control under both irrigated and non-irrigated conditions.

In addition to the data on tomato anthracnose, results were obtained on the severity of blossom-end rot in the various plots and on the overall effect of irrigation on yield of marketable fruit. The number of blossom-end rot affected fruit is presented in Table 3, and the data on marketable yield from irrigated and non-irrigated plots are presented in Table 4.

The data in Table 3 point out the close relationship between unfavorable conditions of soil moisture and the incidence of blossom-end rot. In the irrigated plots, blossom-end rot was virtually non-existent whereas in the non-irrigated plots the disorder was severe. In addition, it would appear that the application of the fungicides gave a significant reduction in the amount of blossom-end rot in the non-irrigated plots. It is possible that the metallic zinc or manganese could play a part directly or indirectly in this influence since other elements such as calcium have been shown to have a similar response (1); or, it may have been the additional moisture applied as spray.

As was expected, the control of both anthracnose and blossom-end rot in this test was reflected in part in the marketable yield. Four irrigations, beginning during bloom and continuing weekly, caused a significant increase in marketable yield from irrigated plots as compared to that from non-irrigated plots. Both fungicides used gave significant increases in yield over the untreated rows under both irrigated and non-irrigated conditions.

Data were obtained also on the occurrence of soil rots due to Pythium spp. and Rhizoctonia solani. The results are presented in Table 5.

The data in Table 5 indicate that under conditions of high soil moisture, tomato fruit rots caused by Rhizoctonia solani and Pythium spp. may be expected to increase in severity. Rhizoctonia rot was much more prevalent than Pythium rot. Only a small percentage of the total fruit harvested were infected by Pythium spp. It is of importance to note that no control was obtained of either rot by the use of fungicides.

DISCUSSION AND CONCLUSIONS

The data obtained in this field test on the influence of irrigation on the development of various diseases of tomato indicate that overhead irrigation will increase the incidence of tomato anthracnose (Colletotrichum phomoides) and fruit rot (due primarily to Rhizoctonia solani) whereas the incidence of blossom-end rot will be reduced. Under non-irrigated conditions, the fungicides used (maneb and zineb) gave essentially the same degree of control of tomato anthracnose but under irrigated conditions maneb gave better control of this disease than did zineb. Neither material afforded control of the soil rots, but there was an indication that they had some influence on the amount of blossom-end rot that occurred in sprayed rows in the non-irrigated plots.

The most important conclusion to be drawn from the test is that different fungicides may behave differently with respect to control of a given disease under irrigated conditions. Thus, materials suggested for use under irrigated conditions must be evaluated under such conditions.

Information is also needed on the timing of applications and on the relative effectiveness of sprays and dusts under irrigated conditions. In addition, it will be necessary to determine what other organisms on tomatoes, as well as other crops, are influenced by irrigation to an extent warranting more detailed investigations as to proper control materials, time of application, and methods of application.

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HUMIDITY REQUIREMENTS OF FOLIAGE PATHOGENS

C. E. Yarwood

ABSTRACT

Foliage pathogens are classified into four types on the basis of their requirements for atmospheric humidity at different stages in their life cycles. These are the anthracnose type in which high humidity is necessary during inoculation, incubation, and sporulation, but not during infection; the downy mildew type in which high humidity is necessary during incubation and sporulation; the rust type in which high humidity is necessary only during incubation; and the powdery mildew and virus type in which high humidity is not necessary at any stage in the life cycle.

Different pathogens have different humidity requirements at the same stage of disease development and the same pathogen may have different humidity requirements at different stages of disease development. Many, if not all, foliage pathogens can be grouped into the four types illustrated diagrammatically in Figure 1. The stages indicated are those undergone by most fungus pathogens and the same terminology can be applied with little difficulty to virus and bacterial pathogens.

The terminology of the stages of disease used here is adapted from Whetzel (16). Inoculation is the transfer of inoculum from its point of production to an infection court. This is sometimes called dissemination, but dissemination is a more inclusive term. Incubation, including germination and penetration, is the development of the pathogen from inoculation until it establishes nutritive relations with the host. Incubation as used here is sometimes included in the term infection as used by others (8). Infection as used here includes all development of the pathogen on the living host after incubation. As used here, sporulation is the formation of spores, or other reproductive units, and may occur during or after infection. The product of sporulation constitutes the inoculum and this completes the life cycle, These same stages are given as transfer, entrance, multiplication, and exit by Riker and Riker (10).

In Figure 1, humidity requirements are given as high or low but quantitative differences within these extremes are indicated. High humidity requirement means that the process indicated proceeds only under conditions of high humidity. Low humidity requirement means that the process may proceed at low humidity but not that low humidity is essential for the process. Quantitative values are not given, but it is believed that the upper limit of the graph should represent about 100 percent and the lower limit about 0 percent relative humidity. Brief descriptions of the types follow.

Anthracnose Type: High humidity is necessary during inoculation, incubation, and sporulation, but not during infection. Conidia are borne imbedded in a matrix in acervuli, and are released by water, especially by splashing rain. Information on the relation of humidity to inoculation, incubation, and infection for Colletotrichum lindemuthianum on bean is given by Edgerton (6).

No quantitative information on the relation of humidity to sporulation of the bean anthracnose organism was found. Bountiful beans were inoculated with Colletotrichum conidia by touching the centers of opposite primary leaves with a brush dipped in a suspension of conidia. Soon after symptoms appeared (5 days after inoculation) the leaves were washed with water to remove surface spores and the plants were placed on a rotating turntable. One leaf of each plant was exposed to the greenhouse environment and the opposite attached leaf was placed at night in a moist chamber, consisting of a pint jar with a notch to accomodate the petiole. The temperature in the greenhouse during this test ranged from 18°C at night to 26° during the day and the relative humidity from 43 percent during the day to 85 percent at night. To measure sporulation, the conidia were washed from the leaves at about 8 a.m., diluted to known volume, and the total number of spores in 0.01 ml aliquots were counted. From this the total number of spores per leaf was determined. The average number of spores per leaf per day from two or more leaves for each determination (not necessarily the same leaves for successive determinations) for the dry (greenhouse) and humid (moist chamber) environments, respectively, was 16,000 and 40,000 at 5 days; 4,000 and 16,000 at 6 days; 4,000 and 55,000 at 7 days; and 1000 and 113,000

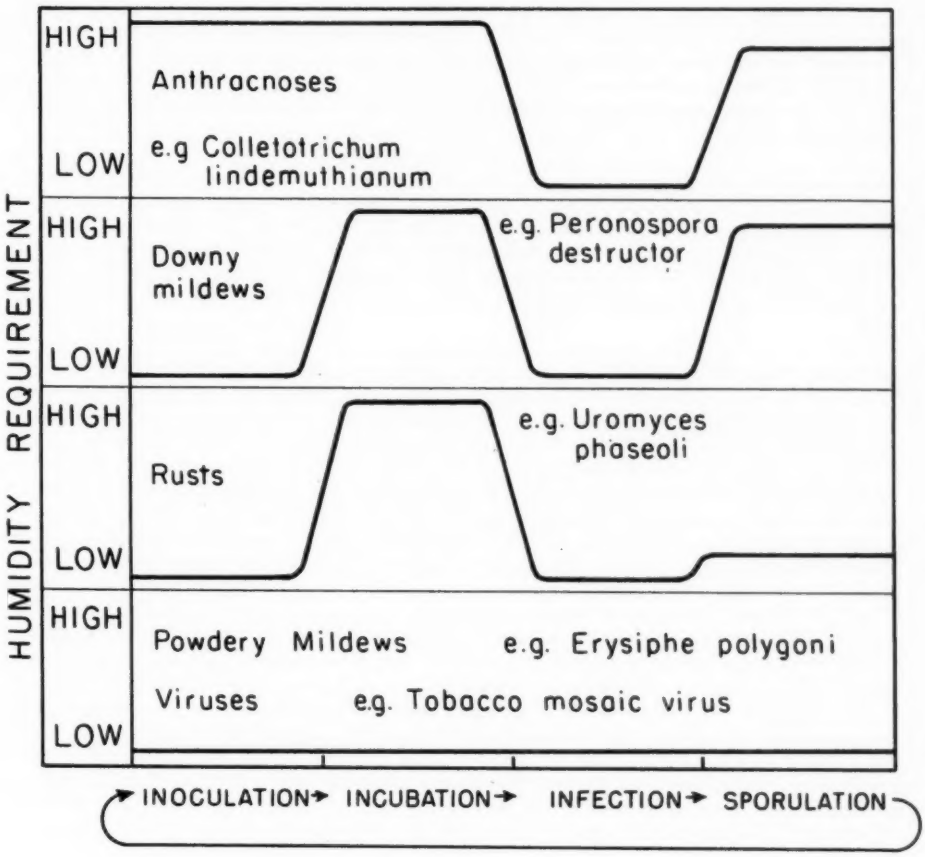

FIGURE 1. Diagrammatic representation of humidity requirements of certain groups of plant pathogens.

at 8 days. The large number of spores on the leaves in the dry environment at 5 days after inoculation was unexpected, but the requirement of high humidity for luxuriant sporulation is clear. On leaf and stem lesions in the still greenhouse environment spores were produced in large numbers, but less abundantly than in moist chambers.

Most acervulus-producing fungi are believed to correspond to C. lindemuthianum in their humidity requirements, though specific data can not be cited. Species of Septoria and other pycnidium-producing fungi also require free water for inoculation and incubation but do not require such high humidity for sporulation as does Colletotrichum. At least three species of Fusicladium require high humidity during inoculation, incubation, and sporulation (Saccas 12) and therefore correspond to the anthracnose type. Many others among the Moniliales probably belong to this group but specific information has not been found.

Several, if not most, bacterial infections of leaves are believed to correspond to the anthracnose type in their humidity requirements. The necessity of free water for inoculation and incubation of angular leaf spot of cotton (Xanthomonas malvacearum) is pointed out by Faul-

wetter (7). The favorable effect of high humidity on the exudation of the bacteria to the surfaces of leaves (sporulation) is indicated by Rolfs (11). That high atmospheric humidity is unnecessary for the intercellular development of bacterial pathogens is implied but not clearly stated by the above and other investigators. That high intercellular humidity is essential for the intercellular development of fire blight bacteria (Erwinia amylovora) is emphasized by Shaw (13), but it is well realized that atmospheric humidity may be relatively low when intercellular humidity is high.

Downy Mildew Type: High humidity is necessary during the incubation and sporulation stages but not during the inoculation stage. Peronospora destructor is a good example (Yarwood, 18). Here, and presumably in all other downy mildews, the humidity requirement is less during sporulation than during incubation. It is believed that many other fungi, such as Cercospora and Botrytis, will be found to correspond to downy mildews in their humidity requirements, but specific information on all phases of the disease cycle for the same fungus has not been found.

Rust Type: For the uredinial stage of rusts, high humidity is necessary only during incubation, but high humidity may favor sporulation. It is well known that high humidity during dissemination (inoculation) and infection is unnecessary, but the fact is poorly documented. It is clearly implied but not specifically stated by Ward (15), Craigie (4), Arthur (1), and Chester (3). The requirement of high humidity for incubation is well known and well documented and is emphasized by Arthur and Chester. With bean rust this period is about 10 hours (Yarwood, 19). It is well known that high external humidity is not necessary for the intercellular development of rusts, but the fact is poorly documented. With bean rust, (Uromyces phaseoli), plants incubated in a moist chamber for about 14 hours after inoculation and then exposed in the greenhouse environment with no special provisions for high humidity have normally shown pustules in 6 days under the driest conditions tested. No requirement of high humidity for sporulation is indicated by most investigators, but increased sporulation of asparagus rust (Puccinia asparagi) and wheat rust at high humidity has been noted by Smith (14) and Manners (9), respectively. In the present study, rusted bean leaves were held in the greenhouse environment and in moist chambers as described above for anthracnose. In 8 pairs of observations the average number of spores per uredinial pustule per 14-hour overnight period was 18 and 31 for 7-day infections, 310 and 392 for 9-day infections, and 3600 and 6000 for 10-day infections, in the greenhouse and moist chamber environments, respectively. The average increase in spore production due to high humidity was therefore only 52 percent for bean rust as compared to 3230 percent for bean anthrancose.

Powdery Mildew and Virus Type: High humidity is not necessary at any time for the development of these foliage pathogens. The tolerance of powdery mildews to low humidity has been emphasized by Brodie (2), Delp (5), and Yarwood (17). The deleterious action of water during inoculation and incubation of certain viruses has been reported by Yarwood (20). It is well known that infection by viruses proceeds normally in dry environments, though quantitative comparisons of virus increase in dry and humid environments have not been found. While there is no sporulation, as such, of viruses, the intracellular formation of new virus units may take place under conditions of low atmospheric humidity.

DISCUSSION

The writer believes that most foliage infections can be classified into one of the four types indicated, but there will be many variations and inter-grading forms. Not enough information is available to classify many pathogens into their proper category. Most pathogens, except powdery mildews and viruses, are presumed to require high humidity or free water during incubation, but most do not require high humidity during infection as defined here. Differences between pathogens are believed to be principally in their humidity requirements during inoculation and sporulation. Most of the information assembled here is not new, but the classification of this information by types of pathogens, and its diagrammatic representation, is deemed useful.

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THE OREGON STRAWBERRY PLANT PROPAGATION CENTER[1]

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Nuclear propagating stocks of 19 strawberry varieties and selected seedlings apparently free from diseases, insects, and nematodes are now available for commercial strawberry plant propagators from the Strawberry Plant Propagation Center at Corvallis, operated jointly by the Oregon State College and the U. S. Department of Agriculture. In addition, planting stocks of 15 promising selected seedlings and 13 named varieties including the virus indicator plant Fragaria vesca, East Malling strain, will be available for testing and increase in 1956.

Production of scientifically pedigreed strawberry plants is comparatively new. Until King (1) showed in 1939 that hyper-sensitive species such as F. vesca could be used as indicators of virus infection, all certification was necessarily based on visible symptoms in varieties that often did not develop symptoms of infection or which did so very slowly. Mild and latent virus could not be detected and the symptoms of more severe ones frequently were masked by unfavorable climatic conditions or by application of large amounts of nitrogen fertilizer.

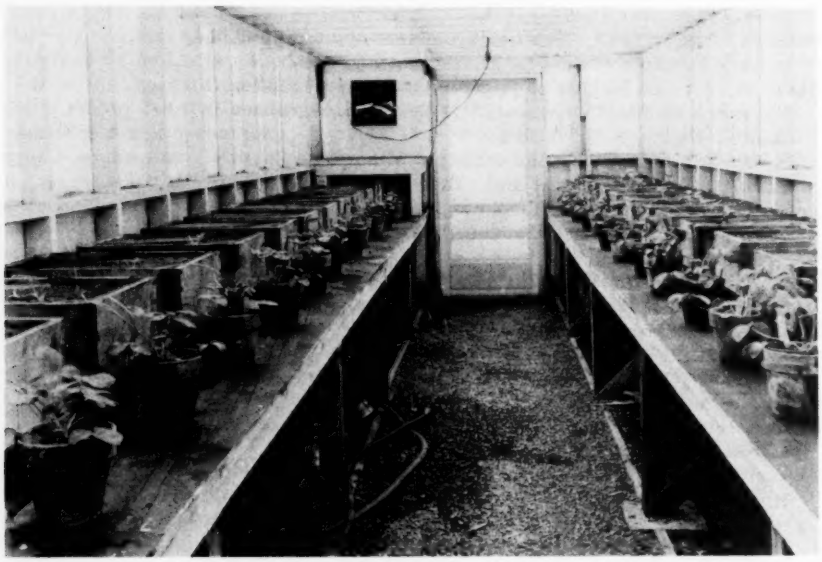

FIGURE 1. View through part of the Oregon Strawberry Plant Propagation Center early in the summer, with runner plant production just starting. Photographs by H. H. Millsap

[1] Approved for publication by the Director of the Oregon Agricultural Experiment Station and by the Chief of the Horticultural Crops Research Branch, Agricultural Research Service, U. S. Department of Agriculture. Contribution of the Departments of Botany and Plant Pathology and Horticulture of the Oregon Agricultural Experiment Station and the Horticultural Crops Research Branch, Agricultural Research Service, U. S. Department of Agriculture.

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Indexing of strawberry propagating stocks was started by United States Department of Agriculture pathologists at Oregon State College in 1950, and by 1953 plants were available which were substantially virus-free (2, 4). This event was hailed as a milestone in the improvement of certified strawberry stocks. Two problems immediately arose, however, to plague both research workers and propagators. How can the plants be kept virus-free, and how can the inadvertent movement of other pathogens in or on the virus-free plants be prevented?

Absolute protection against insect transmission of virus diseases is possible only when the plants are grown in an insect-proof cage or building (Fig. 1). By growing plants in a screenhouse or a well-ventilated screened glasshouse, and keeping them protected by dust or sprays, the plants can be kept virus-free for considerable periods of time.

Practically all soils in the vicinity of the Oregon Experiment Station are infested with the red-stele fungus (Phytophthora fragariae Hickman). Furthermore, all hybrid plants must be tested for resistance to this disease by growing them in infested soil. Thus, all strawberry plants from that area are likely to have been exposed to this disease. The problem of detection of the red-stele fungus is particularly difficult in resistant or immune varieties which do not show symptoms of disease but may still serve as carriers of the fungus.

Chance dissemination of the causal fungus would be of no great concern if all strawberry varieties grown in an area were resistant to the disease, and if there were not distinct physiologic races of the fungus in different areas. Since much of the commercial crop of strawberries is still produced on varieties that are susceptible to most of the known races of the fungus, it is important that nuclear stocks free from the pathogen be developed and maintained, and that only the fungus-free plants be released to growers or shipped from one area to another.

At Oregon State College, a method has been developed for producing strawberry plants free from red-stele even when the parent plant is known to be infected (5). Plants in the propagation center are potted in sterilized soil and pots early in the spring and given applications of fertilizer rich in nitrogen and phosphorus. Before the potted plants produce runners, propagating flats are prepared as follows: deep flats having 1/6 inch mesh hardware cloth bottoms are filled with a mixture of equal parts washed mason's sand, sawdust, and sandy loam and are steam sterilized at 12 pounds pressure for 4 hours. This mixture permits adequate drainage and the runner plants produce luxuriant root systems in the loose sandy soil. The flats are placed on sterilized bricks so that they are several inches above the soil in the pots to prevent splashing of soil particles or oospores of the red-stele fungus into the propagating flats. As soon as the runners appear, they are carried upward and tied to bamboo stakes or wires so that they do not come into contact with the soil in which the mother plant is growing. When the runners are long enough to reach the soil in the flats, they are pegged down with wire staples until the first runner plants are rooted (Fig. 2). The fungus does not invade the crowns or runners and the runner plants are free from red-stele infection.

In order to supply growers of certified strawberry plants with disease-free nuclear stocks, the Oregon Strawberry Plant Propagation Center was started in the spring of 1954, using part of a screenhouse built by the U. S. Department of Agriculture for strawberry virus research. Fifty-five potted plants, each selected as virus-free, were subjected to inspections and regulations more rigid than those for certified stocks belonging to individual Oregon growers.

Only virus and pest-free plants are allowed in the Center. All pots, flats, soil, and frames must be steam sterilized, and after each use they must again be sterilized before they can be returned to the Center. The Center must be sprayed, dusted, or fumigated at regular intervals to keep it free from insects and mites. As a further precaution, the screenhouse is kept locked and only authorized personnel are admitted.

In the past the first two runners produced by each plant have been grafted to a healthy, latent virus-free seedling indicator Fragaria vesca (East Malling Strain) to double check its freedom from virus infection, and all additional runners have been trained upward and pegged down in the flats of sterilized soil. In the future the first runner plant will be rooted in a separate pot, removed to a different greenhouse and grafted to an F. vesca plant carrying latent virus (3). The second runner will be grafted to a healthy, latent virus-free F. vesca plant, and all additional runners will be pegged down in the raised flats of sterilized soil. Two petioles from each mother plant will be plated to check for Verticillium wilt.

At least four inspections are made during the year. The first three, made in July, August, and October, are to check on diseases caused by viruses, fungi, and nematodes. Plants found to be infected with viruses, Verticillium, black root, nematodes, or such local infections as mildew or gray mold, are removed from the Center immediately.

Because red-stele can be detected only after an extended period of cool wet weather, inspec-

FIGURE 2. Strawberry plant propagation technique, showing (Right to Left) virus indicator plant Fragaria vesca runner grafted to mother plant, and first runner plant pegged down in raised flat of sterilized soil. Metal tag on flat bears name or number of the mother plant.

tions for this disease are made in the spring, and plants are released from the Propagation Center only during the period from February 15 to May 1. It is impossible to determine whether the red-stele fungus is present by examining the runner plants or resistant or immune varieties or hybrids: therefore, two susceptible Fragaria vesca plants are planted in each flat in October and examined the following spring for red-stele infection.

In the first year of operation, 15 plant units passed all certification inspections and 3,008 runner plants were made available for sale to propagators or for distribution to cooperating individuals and institutions. In 1955, the Center continued in the same limited space and 37 plant units passed all certification inspections.

However, with increased emphasis being placed on the breeding and testing of new strawberry varieties the present facilities are inadequate to produce the rather large numbers of plants needed. Plans are now taking shape for a greatly expanded program beginning in 1956. A screened and ventilated glasshouse space measuring 70 x 30 feet is being prepared for the Strawberry Plant Propagation Center and in the spring of 1957 adequate numbers of plants free from some of our most troublesome diseases and pests should be available.

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OREGON AGRICULTURAL EXPERIMENT STATION, AND THE HORTICULTURAL CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, U. S. DEPARTMENT OF AGRICULTURE

WILDFIRE CONTROL STUDIES IN BURLEY TOBACCO PLANT BEDS IN NORTH CAROLINA IN 1955

Luther Shaw and George W. Thorne, Jr.

Summary

Different formulations of streptomycin sprays and dusts and Terramycin sprays were tested along with standard copper drench treatments for wildfire (Pseudomonas tabaci) control on inoculated burley tobacco seedlings in the plant bed. Spray treatments with streptomycin at a concentration of 200 ppm and dust treatments with the same material at concentrations ranging from 1000 to 4000 ppm gave good to excellent wildfire control when four applications were made at 1-week intervals beginning immediately after inoculation of the plants with the wildfire pathogen. Four treatment schedules with streptomycin sprays at rates of 5 and 10 gallons of the mixture per 100 square yards at each application were compared and were found to be equally effective in wildfire control. Three applications of streptomycin spray beginning 7 days after inoculation of the plants was equally as effective in wildfire control as four applications beginning immediately after inoculation.

Spray treatments with Terramycin and standard copper drench treatments were substantially less effective in wildfire control than the spray and dust formulations with streptomycin tested.

These results are in general agreement with those reported by other investigators (1, 2, 3).

INTRODUCTION

Wildfire (Pseudomonas tabaci) is one of the most destructive plant bed diseases of burley tobacco. In addition to causing loss of plants in the plant bed, infected transplants serve as an important source of inoculum for field epidemics. Effective control of this disease is an important step in the production of this crop. Drench treatments with copper materials (Bordeaux mixture and tribasic copper sulfate) have been used for many years as control treatments with varying degrees of success. More recently a high degree of control of the disease has been obtained in experimental tests from spray and dust treatments with certain antibiotics (1, 2, 3). In order to gain further evaluations of antibiotics in wildfire control, an experiment was conducted on burley tobacco at the Mountain Research Station, Waynesville, North Carolina during the plant bed season of 1955. A brief report of the methods employed and the results obtained follows.

MATERIALS AND METHODS

Twelve treatments including a control were used. The active materials, concentrations, methods of application, rates of application, and number of applications are given in Table 1. A 9 x 100 foot plant bed which was seeded on March 21, 1955, with the wildfire susceptible Kentucky 16 variety of burley tobacco was divided into 3 x 9 foot plots with boards 7 inches wide, thus giving plots with an area of 3 square yards each. The area was then set up for 12 treatments in a randomized block design with two replications of each treatmnet.

All plots were sprayed with a water suspension of P. tabaci between 1 P.M. and 2 P.M. on May 17. The bacterial suspension was prepared by washing the growth from nutrient agar slant cultures in 14-ounce bottles which had been incubated at room temperature for about 21 days. Each culture was washed with 100 cc of water. This suspension was then diluted with 3 parts of water. Two gallons of the suspension so prepared were sprayed onto the plants in the 72 square yards in the test area. At this time the ground was for the most part covered with plant growth, some of the plants having leaves up to 3/4 inch in width. The suspension applied was sufficient to moisten all exposed plant parts.

The first application of all treatments, excepting the two so indicated in Table 1, were made between 2 P.M. and 4 P.M. on May 17. The dust treatments were applied first, before the bacterial inoculum had dried. The spray and drench treatments were applied later, after the bacterial inoculum had dried. The treatments were repeated at 7-day intervals until the desired number of treatments were made (Table 1).

The dusts were applied with a small pump type hand duster. Applications of the spray formulations were made with a hand type bucket pump which developed a nozzle pressure of about 50 pounds. The drench treatments were applied with a 10-quart sprinkling can. The canvas

Table 1. Wildfire control with certain antibiotics and copper treatments in burley tobacco plant beds[a], North Carolina, 1955.

Treatment	Concentration	Method of application	Rate of application per 100 sq. yds.	Number of applications	Disease index June 15
Agrimycin[b] 100	200 ppm	Spray	10 gals.	3[c]	15.4
		Spray	10 gals.	4	17.8
Streptomycin[d]	200 ppm	Spray	10 gals.	4	14.5
		Spray	5 gals.	4	17.2
	1000 ppm	Dust	3 to 4 lbs.	4	22.7
	2000 ppm	Dust	3 to 4 lbs.	4	21.7
	4000 ppm	Dust	3 to 4 lbs.	4	27.6
Terramycin	200 ppm	Spray	10 gals.	4	48.5
	400 ppm	Spray	10 gals.	3[c]	51.2
Bordeaux mixture	3-4-50	Drench	25 gals.	2	34.9
Tribasic copper sulfate	3-100	Drench	25 gals.	2	40.4
None				-	72.3
L.S.D. (.05)					15.7
(.01)					22.2
C.V. (%)					22

[a] See text for methods employed.

[b] This material contained 15.0% streptomycin and 1.5% Terramycin (Chas. Pfizer and Co., Inc.). The concentrations in ppm for this material refer to streptomycin only.

[c] These treatments were started 7 days after the beds were inoculated with the wildfire parasite.

[d] The sulfate form of streptomycin was used in all formulations.

cover was removed from the plant bed while all treatments were being made.

On May 17, the day the experiment was started, the sky was overcast and the humidity was relatively high. No precipitation occurred during the 24 hours following treatment. Subsequent treatments were made in the morning while the plants were still moist with dew.

It should be noted in Table 1 that the first application of one spray treatment with Agrimycin 100 and one with Terramycin was made 1 week after inoculation. These schedules were designed to measure the possible eradicative effects of these materials on the wildfire pathogen after the plants were infected.

Development of wildfire in the test plots was measured in terms of a disease-index. To arrive at the disease index all plants were pulled from each of two 1-square-foot areas selected at random in each plot. Each plant from each 1-square-foot area was then rated on the basis of wildfire severity, using the following scale: 0 -- none, 1 -- slight, 2 -- moderate, 3 -- medium, 4 -- heavy, and 5 -- severe. To calculate disease indices these readings were given values of 0, 20, 40, 60, 80, and 100, respectively. The average total number of plants examined per treatment was 138.

Weather conditions during the course of this experiment were moderately favorable for wildfire development.

RESULTS

Wildfire lesions developed prominently and abundantly on the foliage of plants in the untreated plots. When the disease readings were made on June 15 considerable yellowing, necrosis, dwarfing, and some leaf drop had taken place in these plots. There was little to no evidence of plant kill, however.

Measurements of wildfire severity in all plots, made 29 days after the experiment was started, show that in terms of a disease index all of the chemical treatments applied, except the delayed treatments with Terramycin spray, resulted in highly significant disease control in comparison with the untreated plots (Table 1). In comparisons among the treated plots all of the spray treatments with streptomycin at 200 ppm gave significantly better wildfire control than did similar treatments with Terramycin and copper drenches.

These differences were highly significant except in comparison with Bordeaux mixture drench.

The results from the analyses of the disease index readings in this experiment indicated that there were no significant differences among the 5 streptomycin treatments although the spray treatments resulted in lower average disease index readings. The data further indicated that the streptomycin dusts were significantly better than the Terramycin sprays in controlling wildfire.

Four applications of streptomycin dust using concentrations of 1,000 and 2,000 ppm were significantly more effective in wildfire control than two drench applications of tribasic copper sulphate using a concentration of 3-100. The data also indicated that there were no significant differences among the three streptomycin dust treatments and the Bordeaux mixture drench as methods for use in controlling wildfire. The inferences based on the analysis of these data are confined to the particular conditions under which the trial was conducted, as well as the experimental materials used.

Of particular interest in this test is the fact that three applications of streptomycin spray (Agrimycin 100) beginning one week after inoculation were equally as effective in wildfire control as four applications beginning immediately after inoculation. This was also true with Terramycin although this material was not so effective in wildfire control as streptomycin.

Four applications of streptomycin spray (200 ppm) were equally effective in wildfire control when applied at rates of 5 and 10 gallons of the mixture per 100 square yards per application.

Blue mold (Peronospora tabacina) did not develop in this test.

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FIELD CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE AND NORTH CAROLINA EXPERIMENT STATION, COOPERATING (MOUNTAIN RESEARCH STATION, WAYNESVILLE, NORTH CAROLINA).

LOCALIZED SYSTEMIC ACTIVITY OF GRISEOFULVIN IN THE CONTROL OF ALTERNARIA BLIGHT OF TOMATO

D. Davis and J. W. Rothrock

Summary

Griseofulvin effectively reduces Alternaria blight of tomato in greenhouse tests when applied as a localized systemic agent. Disease is inhibited when griseofulvin is sprayed on the upper leaf surface and inoculation occurs on the lower leaf surface or vice versa. The effect of griseofulvin persists in the tissues, since plants inoculated 7 days after treatment developed considerably less disease than the controls. The practical implication of the persistent and localized systemic activity of griseofulvin is discussed.

INTRODUCTION

Griseofulvin, a fungal antibiotic isolated from Penicillium griseofulvin by Oxford and co-workers (10) and Brian and co-workers (4, 5) possesses excellent germicidal activity upon a broad spectrum of fungi in vitro (1). When applied to the roots of plants grown in nutrient solution or sand culture it protected the foliage against powdery mildew of barley (2), Botrytis cinerea on lettuce (2, 6) and Alternaria blight of tomato (6, 11). Since Jefferys (8) noted that griseofulvin was degraded in soil, Brian (3) suggested that its potential value might be reduced. The translocative properties of griseofulvin, when applied to the foliage, was investigated by Stubbs (11). In his experiments all but one or two leaves on a given plant were sprayed with griseofulvin. Resistance to disease was then examined in the unsprayed leaves. Stubbs (11) concluded that leaf application was less effective than root absorption in the systemic control of Alternaria blight. Glaxo Laboratories, Ltd. have made further studies confirming the absorption and translocation when griseofulvin is applied to foliage. We are indebted to Glaxo for the generous supply of griseofulvin made available to Merck & Co., Inc. for experimental use and cooperative field trials in 1956.

The present investigation is designed to study the potentialities of griseofulvin as a localized systemic fungicide. Preliminary data on (a) movement from the upper to the lower leaf surface and vice versa, (b) dosage-response and (c) persistence in the tissues was obtained with a view to evaluating its possibilities in the practical control of fungal diseases in the field.

METHODS AND MATERIALS

The Alternaria blight assay developed by McCallan and Wellman (9) was followed in these experiments. Only the area of treatment in relation to the area of inoculation was varied. In all instances, the point of inoculation with spores of Alternaria solani (Ell. & G. Martin) Sor. was on the leaf surface opposite to the leaf surface sprayed with griseofulvin. Griseofulvin was prepared for spraying by dissolving 1 gm in 10 ml of dimethylformamide and 2 ml of Tween 20. The solution was brought to 1000 ml with distilled water.

RESULTS

The data in Table 1 demonstrate that griseofulvin activity is transmitted from the upper surface of the leaf to the lower surface within a 24-hour period. A minimum of approximately 90 percent decrease in Alternaria blight, at all concentrations tested, indicated that griseofulvin is supra-optimal from 125 to 1000 ppm. Extrapolation of the data in Table 1 suggest that LD_{50} values should be considerably below 125 ppm.

Of particular significance is the persistence of griseofulvin (or persistence of response to griseofulvin treatment) in the tissues of tomato leaves. The data in Table 2 show that the effectiveness of griseofulvin is not diminished when inoculation occurs 2 days after treatment. Considerable activity is observed even when inoculation occurs 7 days after treatment. The data in Table 2 likewise indicate that griseofulvin is highly effective whether applied to the lower or upper leaf surface.

It will be noted that the controls inoculated on the upper surface are consistently less diseased than those inoculated on the lower surface (Table 2). This is probably due to the greater washing off of the spores from the upper leaf surface than from the lower leaf surface while the plants are incubating in the moist chamber. Another effect noted was the greater percentage

Table 1. The localized systemic activity of various levels of griseofulvin on Alternaria blight of tomato.

Treatment[a]	Conc., ppm.	Disease Index[b]
Griseofulvin	1000	2
"	500	8
"	250	10
"	125	11
Control	-	96

[a] Inoculated 24 hours after treatment.

[b] Disease index is a measure of the percentage necrotic area on the inoculated leaves. All the leaves on each plant were inoculated. Six plants were used per treatment. The plants had 5 leaves when treatment was initially applied.

decrease in disease of the griseofulvin-treated plants sprayed on the lower leaf surface and inoculated on the upper surface as compared with the reverse sequence (Table 2). This difference may be attributed to the phenomenon observed by Dimond, et al. (7), on the relation between inoculum potential and fungicidal activity in slide germination tests. They found that inoculum potential is inversely proportional to fungicidal activity. This may have a direct bearing on extrapolating greenhouse data to field tests. Under field conditions, the inoculum potential is considerably lower than in our greenhouse tests. Therefore, it might be expected that griseofulvin would be more active under field conditions, if the importance of inoculum potential out-weighs the other factors which might tend to favor griseofulvin in the greenhouse over the field.

DISCUSSION

The implications of this investigation are that the foliage application of a localized systemic agent, such as griseofulvin, while less advantageous than a highly translocatable systemic, may offer considerable practical improvement over the conventional protectant fungicides.

When applied as a foliage spray in the field, griseofulvin might have the following advantages over a conventional protectant: (a) There would be less need for 100 percent coverage. Direct application to the lower leaf surface might be unnecessary. (b) There would be less of a problem with wash-off. A heavy rain within 24 hours after application would not necessitate an immediate re-spraying. (c) A greater lapse between spray applications; therefore fewer sprayings in a growing season. The persistence of griseofulvin activity in the tissues for at least a week is indicated by our greenhouse tests.

Table 2. The localized systemic activity and persistance of griseofulvin (1000 ppm) in the control of Alternaria blight of tomato.

Treatment	Time of Inoc. (In days after treatment)	Surface of Leaf Inoculated[a]		Disease Index[b]	
		Lower	Upper	Avg.	% of control
Control	0 (2 hrs.)		✓	30	
Griseofulvin	0 (2 hrs.)		✓	1	3
Control	0 (2 hrs.)	✓		100	
Griseofulvin	0 (2 hrs.)	✓		9	9
Control	1		✓	22	
Griseofulvin	1		✓	1	4
Control	1	✓		100	
Griseofulvin	1	✓		17	17
Control	2		✓	17	
Griseofulvin	2		✓	1	4
Control	2	✓		53	
Griseofulvin	2	✓		3	5
Control	7		✓	60	
Griseofulvin	7		✓	18	30
Control	7	✓		98	
Griseofulvin	7	✓		37	37

[a] Leaves treated on the surface opposite to the surface inoculated.

[b] Disease index is a measure of the percentage necrotic area on the inoculated leaves. All the leaves on each plant were inoculated. Three plants were used per treatment. The plant had 5 leaves when treatment was initially applied.

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AVAILABILITY OF STREPTOMYCIN IN DUST FORMULATIONS

Peter A. Ark and Eugene M. Wilson[1]

Summary

Pyrophyllites (Pyrax ABB and Nuclay), hydrated lime, sulfur, calcium and magnesium carbonates serve as excellent dust carriers of streptomycin, releasing it readily when in contact with water. Fuller's earth, talc, frinite, attaclay, and Taco clay bind streptomycin and render it unavailable when mixed with water.

The high pH of hydrated lime and of lime-sulfur (1% and2%) does not affect the availability of streptomycin when the latter is blended with those materials.

Both pyrophyllite- and hydrated lime-streptomycin formulations gave good control of angular leaf spot of cucumber (Pseudomonas lachrymans) where dusted on both sides of the leaf followed by mechanical inoculation.

Streptomycin treatment of plants has been shown to be of definite benefit for a number of plant diseases (1, 2, 3, 5, 6). From the standpoint of economics, streptomycin is considered to be more costly than other agricultural chemicals capable of giving a measurable degree of protection from plant diseases. However, streptomycin is known to be safe and slightly systemic. Therefore, it was desirable to develop dust formulations that would lower the cost of the treatment where, because of climatic conditions, several applications might be required.

In an attempt along this line made in 1953 (1), it became apparent that bentonite dust as a carrier was not satisfactory because it adsorbed streptomycin almost irreversibly, thus rendering it less effective than wettable forms. To find a more suitable carrier for streptomycin, tests were made with a number of diluents most commonly used in formulating fungicides and insecticides. This paper deals with the studies made with Fuller's earth, frinite, attaclay, Taco clay, talc, Pyrax ABB (pyrophyllite), Nuclay (pyrophyllite), hydrated lime, and sulfur in combination with crude streptomycin,

The dust formulations were prepared by mechanically blending the carrier and the streptomycin in a Raymond laboratory mill. All formulations contained 1000 ppm of streptomycin. Determination of the activity of the dusts was made after various intervals to determine their keeping qualities.

In order to measure the amount of streptomycin released by a given formulation, 1 gram was placed in a centrifuge tube in 10 ml of distilled water, shaken vigorously (by hand) for about 5 minutes and then placed in a Servell centrifuge for spinning for 20 minutes at about 8000 r.p.m. The supernatant fluid was collected in a small beaker and subsequently absorbed by the S and S #740-E bioassaying filter paper discs. The discs were placed in sterile petri dishes and dried at 40° C for one or two hours after which they were placed on poured plates of clean potato-dextrose-peptone agar medium seeded with a 24-hour old culture of Erwinia amylovora and surface-dried for 20 minutes at 40° C. Plates so prepared were incubated, in an inverted position, for 24 hours at 28° C and the inhibition zones around the discs were measured (diameters) in millimeters. Check consisted of comparable discs dipped in a solution of crude streptomycin containing 100 ppm.

Of the materials reported in this paper the Fuller's earth, talc, attaclay, frinite, and Taco clay were found to be unsuitable for streptomycin because of its irreversible union with them. Addition of 1% K_2HPO_4 helped to release the streptomycin from all these carriers except the talc.

The pyrophyllites Pyrax ABB and Nuclay, hydrated lime, and sulfur (micronized) proved to be excellent dust carriers for streptomycin. Apparently they released streptomycin almost completely when they were eluted with distilled water, and they did not diminish the activity of streptomycin even after many months of storage. Thus, the amount of the streptomycin released by Pyrax ABB, Nuclay, and sulfur was about the same after 12 months as it was immediately after preparation (Table 1).

In the hydrated lime-streptomycin formulation, streptomycin remained in the same concentration 2 months after its preparation, the time during which it was under study. It appears that the stability of the streptomycin was not affected by the high pH value (12.7) of the lime,

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Table 1. Release of streptomycin from different streptomycin-dust formulations after different intervals of storage. Washings for bioassaying contained 100 ppm of streptomycin.

Dust formulations	Date of preparation	pH	Dates of bioassaying							
			Feb. 2, 1955	March 6, 1955	June 6, 1955	Sept. 6, 1955	Dec. 4, 1955	Dec. 29, 1955	Jan. 13, 1956	Jan. 24, 1956
			Size of inhibition zones: dia. - mms.							
Pyrax ABB Strept., 1000 ppm	2/1/55	6.5	24	24	23	24	24	23	24	24
Nuclay Strept., 1000 ppm	3/5/55	8.9	--	23	23	24	24	24	23	23
Sulfur Strept., 1000 ppm	3/1/55	6.9	--	24	24	24	23	23	24	24
Hydrated lime Strept., 1000 ppm	12/2/55	12.5	--	--	--	--	23	22	22	23
Check (wettable Strept., 100 ppm)	---	6.9	24	24	24	23	24	24	23	24

contrary to some earlier reports in the literature on the adverse effects of high pH on streptomycin. That alkalinity does not inactivate streptomycin is apparent from its ready demonstration in 1% and 2% lime sulfur, with pH of 10.5 and 10.7, respectively. Streptomycin solutions containing 1000 ppm prepared in 1% and 2% lime sulfur on December 15, 1955 and tested on December 15 and December 30, 1955, January 13 and January 24, 1956, showed the same zones of inhibition as the check prepared in distilled water. Checks made of lime sulfur (1% and 2%) without streptomycin had no inhibitory effect on the test organism. However, the activity of streptomycin prepared in a straight (undiluted) lime sulfur, the pH of which is 11.0, was demonstratable only at the time of preparation and 24 hours after preparation and not later. Whether this is due to inactivation or merely adsorption cannot be answered at this time.

Limited tests with calcium and magnesium carbonates as carriers showed that streptomycin can be safely combined with these diluents, with no inactivation of the antibiotic after 30 days of storage at 24° C in an open container. This may indicate that the addition of either calcium or magnesium carbonates to improve the flow of certain dust formulations would be safe.

The efficacy of the streptomycin-pyrophyllite formulation against fireblight was recently reported by the senior author (2). To ascertain its potentialities against other phytopathogenic bacteria, the cucumber angular leaf spot organism (Pseudomonas lachrymans) was selected. Cucumber plants grown in 5-inch pots, three plants to a pot, were allowed to develop to the six-leaf stage when the tips were cut back. Inoculation was made by gently rubbing the leaves with the fingers contaminated with the bacterial exudate from diseased cucumber leaves. This technique was adopted to simulate the actual manner of spread of the disease by pickers in cucumber fields (4). Preliminary trials indicated that a high degree of infection is produced on cucumbers when inoculation is made by contaminated hands as described above. Only one dusting on both sides of the leaves was made prior to inoculation. Final readings were made one week after the treatment although good symptoms are usually well developed by the fifth day in a greenhouse where the temperature fluctuated from 90° to 65° F and a moderate humidity was maintained by sprinkling greenhouse walks and benches. The results of the three separate tests indicated that, whereas quite heavy infections developed on nontreated inoculated plants, a very small amount of the disease developed in a hydrated lime-streptomycin treated lot and practically no apparent infections in a Nuclay-streptomycin dust lot, while a moderate amount of the disease was present in both wettable streptomycin and copper-lime dust groups.

Bordeaux (6-6-50) treated plants developed a light amount of the disease. It is seen from these limited tests that the streptomycin-pyrophyllite (Nuclay) dust gave considerable reduction of angular leaf spot of cucumber while the streptomycin-lime combination was next most effective. Both streptomycin dust formulations gave better performance than either copper-lime dust or Bordeaux. One spray of wettable streptomycin, 100 ppm, did not give a degree of the control comparable to the streptomycin dusts used in the trials.

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BACTERIAL SPOT OF PEACH (XANTHOMONAS PRUNI) AND ITS CONTROL BY THE USE OF CAPTAN AND STREPTOMYCIN[1]

Robert H. Daines

Bacterial spot of peach, caused by Xanthomonas pruni, has not yet been adequately controlled by the use of chemical sprays. Zinc sulfate and lime, even when applied thoroughly and often, does no more than half a job in controlling this disease, and in New Jersey this mixture so often proves to be phytotoxic that it is of no real value in a spray program here. Captan at 3 pounds has become the standard New Jersey remedy in combating this disease but it also leaves much to be desired. At this concentration leaf lacing or shot holing frequently develops, and some defoliation following its use has been observed. Antibiotics as foliage sprays have shown little promise of effective control of bacterial spot. Dunegan and others reported (1, 2) that Terramycin injected into trees gave protection against this disease, but they also found that this treatment produced chlorotic foliage.

Goodman and Shepard (3) reported beneficial results from foliage sprays of streptomycin in a non-replicated test. In New Jersey, however, bacterial spot so often develops without uniformity in a block that replication of treatments is absolutely necessary for reliable results. Streptomycin has been included in control experiments in New Jersey during the past 4 years. During 3 of these years the disease did not develop in the test area, but in 1955 the disease became severe on the foliage and fruits of nearly all of our sprayed trees (Table 1).

Table 1. Control of bacterial spot of peach with captan and streptomycin foliage sprays.[a]

Treatment used[b]	Fruits Showing Bacterial Spot Percentage
Unsprayed Check	31.6
Sulfur, 5 pounds per 100 gallons	24.8
Captan, 2 pounds	20.0
Captan, 3 pounds	15.3
Captan, 4 pounds	9.6
Captan, 2 pounds, Streptomycin, 150 ppm	7.5
L.S.D. 5%	15.3
L.S.D. 1%	21.1

[a] Constitution variety. (Bridgeton, New Jersey).

[b] Streptomycin sulfate was used in this test. The streptomycin used was supplied by the American Cyanamid Co., and captan by the California Spray Chemical Company.

During the 1955 growing season, trees of the variety Constitution were divided into five blocks with each block consisting of one randomized tree for each treatment. By July 6 considerable bacterial spot had developed in the experimental area, and beginning with that date these trees were sprayed on a 7- to 10-day schedule. During the excessively dry summer season that followed, bacterial spot made little or no progress, but during the hurricane of August 17 infections of both fruit and foliage became numerous. The trees in this test had been sprayed on August 16, just before the winds and heavy rains. On September 7, after the

[1] Paper of the Journal Series, New Jersey Agricultural Experiment Station, Rutgers University, the State University of New Jersey, Department of Plant Pathology, New Brunswick.

late infections on the fruit had developed clearly visible symptoms, records were made of diseased and healthy fruits to compare the various treatments (Table 1). In these records only late infections were considered. It is clear that with increasing concentration captan gave increasing protection against bacterial spot. The outstanding treatments proved to be captan at 4 pounds, and a combination of captan and streptomycin. Streptomycin appeared to give considerable protection, since the captan (2 pounds)-streptomycin combination was significantly better than the check treatment, whereas captan alone (2 pounds) was not. Bacterial spot data were not taken on the foliage but effects appeared to be similar to those for fruits, although smaller.

On August 31, just before the last spray was applied, foliage and fruits from trees (four) that received the streptomycin sprays and from trees that did not receive the antibiotic were picked and taken to Dr. Phillips of Merck & Company, where they were bioassayed to determine if streptomycin had been absorbed by these plant parts. The assays revealed that streptomycin was present at an average concentration of 9 ppm in the foliage juice of washed leaves. The fruits gave no positive test for the antibiotic.

So far as the writer has been able to determine, this is the first time that evidence has been obtained showing penetration of peach foliage by streptomycin. Whether this activity was due to captan or its wetting agent, the pH of the spray film, the variety of peach used in this test, or other factors is not known.

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SULFUR, KARATHANE, AND ACTIDIONE FOR CONTROL OF POWDERY MILDEW OF WHEAT[1]

Willard Crosier and Michael Szkolnik

Summary

Seedlings of Genesee and Yorkwin wheat in greenhouse culture were infected by powdery mildew, Erysiphe graminis tritici E. L. Marchal.

Sprays of Micronized sulfur 3-100 or 6-100 and of Karathane 1-100 controlled mildew whereas Actidione 2ppm or 3ppm reduced it slightly. A combination of Actidione and sulfur was not superior to sulfur alone.

When sprayed on the existing first and second leaves, any of these materials reduced the number of lesions that appeared on the subsequently developing third and fourth leaves.

At a concentration of 3ppm, Actidione caused a marginal discoloration and necrosis of wheat leaves.

During January 1955 wheat seedlings transplanted from open fields to a cool greenhouse became heavily infected with powdery mildew, Erysiphe graminis tritici. Although dusting with sulfur is known to be an effective control measure, spraying with this or other materials seemed more suitable for plants growing in closely spaced rows.

An entire bed of Genesee wheat in the 1-leaf stage was divided into six blocks and two of each sprayed with Actidione 2ppm, Karathane 1-100, or water alone. No wetter was used, but spraying was continued until a thorough uniform coverage of all the foliage was attained. Pots of heavily infected seedlings were placed at 2-foot intervals throughout the six blocks.

The entire greenhouse was warmed to 65°F continuously. The foliage was sprayed with water daily.

In 7 days every non-protected plant appeared to be mildewed. Scattered infections were observed in the Actidione-sprayed blocks while the Karathane-sprayed blocks were still healthy. After 2 days more, the percentage of infected leaves was estimated as: Actidione, 30; Karathane, 5 within 8 inches of inoculum and 0 elsewhere; and check, 100.

The plants were pulled from the seedbed 14 days after spraying. Each leaf was examined for mildew and the infections were counted on every tenth plant. As shown in Table 1, Actidione 2ppm was relatively ineffective whereas Karathane 1-100 afforded good protection to the first or bottom leaves. Only the tips of the second leaves had received any spray, yet both Actidione and Karathane appeared to reduce the number of infections.

Table 1. Powdery mildew on Genesee wheat seedlings.

Spray material[a]	Percent of infected leaves		Number of infections per 100 leaves	
	First	Second	First	Second
Actidione 2ppm	84	86	795	480
Karathane 1-100	26	45	86	225
Check	82	83	1460	810

[a]No wetting agent used.

Yorkwin wheat seedlings grown to the 3-leaf stage in either soil or Terralite were sprayed on February 26 with Actidione 3ppm, Karathane 1-100, Micronized sulfur 3-100, or water alone. Infected foliage was spread on the sprayed plants and between the flats containing the plants.

On March 7, the check plants were noticeably white. About 30 percent of the Actidione-sprayed, 2 percent of the Karathane-sprayed, and none of the sulfur-sprayed plants were infected.

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Table 2. Influence of vigor of Yorkwin wheat plants upon control of powdery mildew.

Spray material[a]	Number of lesions per 100 leaves			
	First leaf		Second leaf	
	Soil	Terralite	Soil	Terralite
Actidione 3ppm	810	101	1277	53
Karathane 1-100	61	88	69	2
Micronized sulfur 6-100	19	12	11	1
Check	2386	1910	2667	2124

[a]Triton X-100 1 drop per liter as wetter.

Table 3. Extent of protection afforded from sprays on first and second leaves.

Spraying material[a]	Average number of lesions on			
	First leaf	Second leaf	Third leaf	Fourth leaf
	Plants growing in soil			
Actidione 3ppm	9.3	12.8	12.0	9.5
Karathane 1-100	0.0	0.6	2.4	0.8
Micronized sulfur 3-100	1.2	1.7	1.6	0.1
Micronized sulfur 6-100	0.0	0.5	2.9	1.4
Micronized sulfur 3-100 + Actidione 3ppm	0.3	1.3	3.6	1.1
Check	26.8	22.7	19.4	11.9
	Plants growing in sand			
Actidione 3ppm	3.1	1.4	1.5	0.3
Karathane 1-100	0.2	0.5	0.2	0.0
Micronized sulfur 3-100	0.0	0.1	0.0	0.0
Micronized sulfur 6-100	0.0	0.0	0.2	0.2
Micronized sulfur 3-100 + Actidione 3ppm	0.2	0.8	1.8	0.4
Check	2.4	3.7	3.0	1.6

[a]Triton X-100 1 drop per liter of all fungicide treatments.

On March 11, 50 of the approximately 490 plants in each flat were examined for mildew lesions. As shown in Table 2, the larger, thriftier plants growing in soil were more heavily infected than those growing in Terralite. This was due in part to the greater leaf area and possibly also to the reduced spray penetration into the mass of the larger plants.

Actidione was noticeably less fungistatic than Karathane or sulfur in controlling mildew on the plants grown in soil. The leaves were sprinkled abundantly during the daily watering, but loss from washing cannot be accountable for the comparative ineffectiveness of Actidione.

In order to determine what effect materials applied to existing leaves would have on the mildew control of subsequently developed leaves, Yorkwin seedlings were sprayed in the 2-leaf stage and held until the 5-leaf stage. The seedlings were grown in flats and 10 of these units were sprayed with Actidione 3ppm, Karathane 1-100, Micronized sulfur 3-100, Micronized sulfur 6-100, Micronized sulfur 3-100 plus Actidione 3 ppm, or water alone. Triton TX-100 1/2 oz.-100 was used as the wetting agent.

Inoculum was supplied by heavily infected plants placed within 12 inches of any sprayed plant. Spores of Erysiphe graminis tritici were continuously present for the 24-day duration of the experiment. During this time the third, fourth, and fifth leaves appeared. The plants were wet daily while watering.

As shown in Table 3, Actidione was consistently inferior to Karathane and sulfur. Actidione was the only material to cause leaf injury, as expressed by marginal discoloration and necrosis. The addition of Actidione to sulfur usually effected a neutral or negative control response.

The protection afforded by any material extended even to leaves formed after application of the spray. This finding suggests that under greenhouse conditions mildew could be controlled without the prompt coverage of each developing leaf.

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REACTIONS OF CERTAIN SPRING BARLEY LINES TO RACE 59A OF STEM RUST[1]

John D. Miller and J. W. Lambert[2]

Abstract

Over 1200 spring barley lines were screened for reactions to race 59A of stem rust as seedlings in the greenhouse and as adult plants in the field. Depending upon the classification used adequate resistance was found. One variety, Black Hulless, CI 666, was found to have outstanding resistance in both the seedling and adult stages. Statistically significant associations were present between the reactions of seedlings and adult plants.

INTRODUCTION

In 1950 in the spring barley breeding nurseries at the University of Minnesota, St. Paul, a high prevalence of susceptible type pustules of stem rust were found on segregating material and lines which were expected to carry stem rust resistance. Subsequent identification disclosed that race 59A of black stem rust, Puccinia graminis tritici, was causing this susceptible reaction. The breakdown of hitherto resistant varieties pointed up the need for additional information on the reaction of barleys to this race of stem rust. This paper reports such studies.

The following information on distribution and prevalence of race 59A of black stem rust was taken from publications by E. C. Stakman et al. (4). Race 59 was present in the United States in uredial collections as early as 1930 and in aecial collections in 1929 (4). It was not found west of the Mississippi until 1944 when it was reported from aecial and uredial collections in Washington (4). In that year the various biotypes of race 59 were reported separately. In 1944 race 59 made up 2.0 percent of all isolates. Race 59A made up 27 percent of all aecial collections of race 59 and 46 percent of all uredial collections of race 59 in 1944. The race continued to be present in the Eastern States and the far West until 1950 when it was found in Minnesota also. Race 59 ranked from fourth to sixth in prevalence for uredial collections during the period 1944-1951 with biotype 59A composing about one-fourth to one-half of the total. During the period 1944-1950 race 59 made up from 2.7 to 20 percent of all aecial isolates with biotype 59A making up about one-fifth of the total for the race. The race has continued to be present in aecial and uredial collections since 1951 but has been confined to areas where barberry is present (4).

The literature on race 59 is limited. Ali (1) reports the behavior of various biotypes including 59A on certain wheat and barley varieties. He found an indication of strains within the established biotypes of race 59B and 59C. Race 59A has been reported to be especially virulent on barley (4).

MATERIALS AND METHODS

Initial exploratory work was started in the summer of 1951 when 1042 varieties from the World Collection of barleys and about 140 varieties from rod-row tests were inoculated with six races of stem rust, namely 15B, 17, 19, 38, 56, and 59A. During the winter of 1951-1952, seedling reactions at a temperature of about 82° F were obtained for 1042 varieties from the World Collection and about 140 varieties from the Minnesota testing program. This same group of varieties was grown to maturity in 1952 in a field nursery inoculated with race 59A.

Seedling tests were made on about 25 seedlings planted in autoclaved soil. Seed was treated

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with a dilute mixture of New Improved Ceresan to inhibit damping off. Inoculation usually was made 7 to 8 days after planting.

Plants to be inoculated were sprayed with water and rubbed to remove the waxy bloom. The plants were again sprayed with water whereupon a spore-talc mixture was applied to each pot by means of an air bulb attached to a glass cyclone chamber (5). Following inoculation the plants were again sprayed and covered with a moist tarpaulin for about 36 hours at 70° F. After this the cover was removed and pots widely spaced on the bench to permit air circulation and to provide maximum light. Supplemental light was provided from 11 p.m. to 8 a.m. A recording thermograph showed that temperatures were maintained about as planned.

Field plots were single 6-foot rows spaced 1 foot apart with 3- or 4-foot alleys between beds. In the alley and around the outside border of the nursery, spreader rows of susceptible barleys were planted about 1 week earlier than the nursery itself. The spreader rows were inoculated at 3- or 4-day intervals with an aqueous spore suspension injected hypodermically at the preboot and boot stage.

Notes were taken for seedlings on the range of pustule type present with the predominant type noted. The prevalence of pustules was noted on a percentage scale. On adult plants notes were taken on the pustule type and percentage rust.

The scale used for seedling notes is a deviation from the one used for wheat and hence is described and illustrated (Fig. 1). The new scale was necessitated by the fact that barley varieties respond to stem rust differently from wheat. Chlorosis tends to be present even in the more susceptible varieties. Furthermore the characteristic type 2 pustule is not common in most barley varieties.

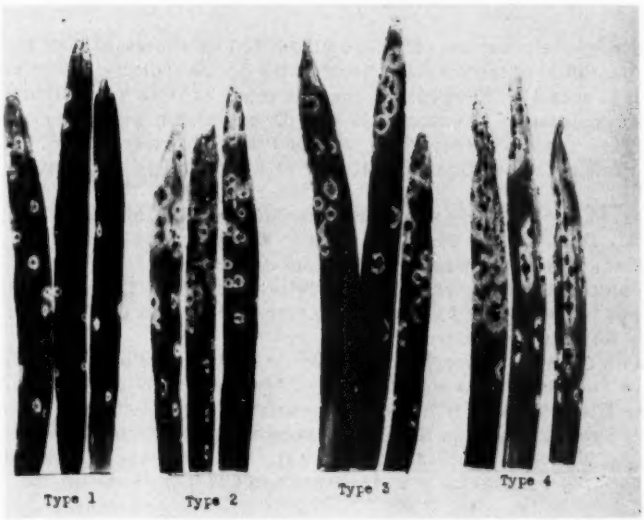

FIGURE 1.
Reaction types used in classifying stem rust infection on barley seedlings.

In the revised scale pustule size is emphasized but in order to preserve as much similarity as possible to the wheat scale the classes are maintained as 0; to 4. The various classes are as follows:

Type 0; -- Characterized by hypersensitive flecks with no visible sporulation present.

Type 1 -- Uredia are small (not over 1 mm) and surrounded by distinct necrotic areas.

Type 2 -- Uredia are larger than type 1 and range up to 2 mm. Chlorosis is usually present but some pustules are surounded by a necrotic area.

Type 3 -- Pustules are moderately large (from 2 to 4 mm) and occasionally coalesce. Uredia are surrounded by chlorotic or necrotic borders.

Type 4 -- Pustules are large (over 4 mm) and often coalesce. Borders are usually chlorotic but sometimes necrotic.

Plus and minus signs were used to delimit pustule sizes more precisely. Notes were taken on the presence of chlorosis or necrosis.

EXPERIMENTAL RESULTS

Sources of Resistance

The adult plant reaction will be considered in this section since it is the most important criterion of stem rust resistance, particularly from the economic point of view. The source of race 59A used for adult and seedling tests was a collection designated by the Federal Rust Laboratory as race 59A Zeil 1A:[3].

Reaction classes for adult plants were established on the combined basis of pustule type and percentage of rust. The resistant class included varieties with resistant or moderately resistant type pustule and/or those with less than 2 percent of rust. The intermediate class included varieties with a moderately susceptible type pustule and/or 2 to 10 percent of rust. The susceptible class included varieties that had susceptible type pustules and more than 10 percent of rust. Use of percentage of rust as a criterion of resistance is based on the assumption that varieties with low percentages of rust even of a susceptible type may offer satisfactory resistance under epidemic conditions from an economic viewpoint.

Two groups of material were grown. The first group comprised the varieties and lines from the Minnesota breeding program in two replications. The second group was a portion of the World Collection of Barleys. Inasmuch as rust was definitely lighter in prevalence in the part of the nursery containing the Minnesota strains, it seems advisable to consider the two groups separately.

Of the 183 lines from the Minnesota program, 65 were classified as resistant. Of these almost all had susceptible type pustules on the stems. None of the commercially grown varieties was classified as resistant (Table 1). However, a large number of lines from hybrids involving commercial lines were resistant. Brandon 1136 and its selections were outstanding. Other hybrid combinations that gave resistant selections included the following: Minn. 601 x Wis. H-35, Mars x Kindred, Brandon 1136 x Kindred, Minn. 601 x Montcalm, and Mars x Montcalm.

Of the common varieties tested the following were intermediate in reaction: Peatland, Chevron, Feebar, Trebi, Tregal, O.A.C. 21, Barbless, Mars, Kindred, and Montcalm. The presence of Montcalm and Barbless which are susceptible to the common races of stem rust in the intermediate class, as well as the normally resistant varieties Peatland, Chevron, and Kindred, indicates that resistance is conditioned by genetic factors other than the T factor found in Peatland, Chevron, and many of their derivatives.

As compared to the Minnesota group, the World Collection of barleys had a much lower percentage of varieties that were resistant to race 59A. Out of 1042 tested 37 were classified as resistant. Only one of these, Black Hulless (CI 666), had resistant type pustules (Table 1). It is perhaps significant that this variety was also highly resistant to race 59A in the seedling stage and also to stem rust of rye, Puccinia graminis secalis (2). This variety originated in China. Of the 1042 varieties 168 were intermediate in reaction and 837 were susceptible (Table 2).

It appears that adequate resistance exists to this race but that the gene or genes governing resistance are not identical with the Peatland or Chevron factor T.

Correlation of Seedling and Adult Plant Reactions

The correlation of reactions of seedlings and reactions of adult plants was made on the two groups of material already described, i.e., the World Collection and Minnesota strains. The reaction classes for adult plants were described in the preceding section. The class limits used for seedlings are as follows: resistant -- type 2 or less; intermediate -- type 2+ or 3-; and susceptible -- type 3 to 4+. Using these scales the Minnesota strains were classified for seedling and adult reactions with the results shown in Table 2. The X^2 for association indicates that there was a fair association of adult and seedling reactions. The resistant or intermediate seedling reaction was particularly effective in selecting against strains which would be susceptible as adults.

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[3] The writers are indebted to the Federal Rust Laboratory, St. Paul, Minnesota for supplying this race of stem rust.

Table 1. Reaction to race 59A of certain barley varieties in the seedling stage and as adult plants.

Variety	C.I.No.	Adult Reaction: Pustule type	Adult Reaction: Prevalence (%)	Seedling Reaction
Kindred	6965	S	3	1-2+cn
Montcalm	7149	S	10	1-4⁻cn
Wis. Barbless	5105	S	8	1-3+cn
Mars	7015	S	7	1+-2+cn
Moore	7251	S	20	2⁻-4⁻cn
Feebar	7260	S	3	1+-3⁻cn
Trebi	936	S	4	1-4cn
Tregal	6359	S	3	1-4⁻cn
O.A.C. 21	1470	S	8	2⁻-4cn
Plains	7250	S	15	1+-3+cn
Brandon 1136	--	S	tr+	1-3⁻cn
Brandon 1136 Sel.I-48-5	9187	S	tr+	1⁻-3+cn
Brandon 1136 Sel.I-48-17	9188	S	tr+	1-3cn
Peatland	5267	S	5	1+-3+cn
Fort	8114	S	10	1-4⁻cn
Chevron	1111	S	3	1-3⁻cn
Wis. x 330-5	9190	S	tr	1-3⁻
Bohemia	32	S	1	2⁻-3⁻
Bavarian	160	S	tr+	1-2+
Prize Prolific	169	S	1	1+
French Chevalier	175	S	1	1-2+
Chevalier II	200	S	tr+	1+-2+
Bohemian	204	S	1	2+-2+
Pride of Dakota	218	S	1	1+-2+
Hanna	226	S	1	1-3⁻
Manchuria	241	MS	1	1-2+
Goldthorpe	327	S	tr	1-2+
Abyssinia	362	S	tr	1-3⁻

Table 1. (Continued)

Variety	C.I.No.	Adult Reaction: Pustule type	Adult Reaction: Prevalence (%)	Seedling Reaction
Nepal	475	S	tr	1+-2
Princess	529	S	1	1-2
Chile Chevalier	659	S	tr	1-3⁻
Black Hulless	666	R	tr⁻	0:-1
Abyssinia	668	S	tr⁺	1-2+
Franconian	680	S	1	1⁻-2+
Baku	709	MS	2	1-2
White Gatami	920	MS	2	1-2
Balton	922	S	1	1-2+
Kite	992	S	tr	1-3⁻
Kurof	1098	S	1	2⁻-3+
Curov	1099	S	1	0:-3+
Tivarmes	1109	S	1	1-3
Abyssinian	1227	S-MS	tr	1-4⁻
Abyssinian	1240	S	tr	1⁻-3⁻
Redfield	1295	S	tr⁺	1-2
Fabian	1458	MR	tr	1⁻-1+
Bucher	1461	S	1	1⁻-2
Purple Nudum	2250	S	1	1-
Abyssinian	2251	MS	tr+	1+-3
Nesbit	2451	MR-S	tr	2+-3
Selca	2452	MS	1	1+-3+
---	3381	MR	tr	1-3+

Table 2. Association of seedling and adult plant reactions of Minnesota strains of barley to race 59A of stem rust.

		No. of Varieties (Adult)			
		R	I	S	Total
No. of Vars. (Seedlings)	R	14	9	0	23
	I	46	85	8	139
	S	5	11	5	21
Total		65	105	13	183

$X^2 = 17.2757$, D. F. = 4, $P = < .01$

Table 3. Association of reactions of seedling and adult plant to race 59 of certain barley strains from the World Collection of barleys.

		No. of Varieties (Adult)			
		R	I	S	Total
No. of Vars. (Seedlings)	R	10	29	29	68
	I	20	105	227	352
	S	7	34	504	545
Total		37	168	760	965

$X^2 = 157.4000$, D. F. = 4, $P = < .001$

The 1042 varieties from the World Collection of barleys were tested in a similar manner with the results shown in Table 3. A high degree of association is indicated by the X^2 value. The intermediate or susceptible reaction was more effective for eliminating lines undesirable from the standpoint of their rust reaction.

DISCUSSION

The chief economic importance of race 59 and biotype 59A of stem rust seems to be on barley (6). The limited acreage of barley plus its earliness as compared to spring wheat together with the resistance to the race found in wheat will probably limit the buildup of the race enough to prevent serious economic losses. However, there have been enough cases on record of the sudden buildup of an old or new race to epidemic proportions to indicate that it is unsafe to ignore any race, especially one which has demonstrated such virulence as race 59A in the

field and greenhouse.

Although a few varieties, in particular Black Hulless (C.I. 666) appeared to be consistently resistant to Race 59A, the lack of clearcut association between greenhouse and field tests is of some concern. The failure to obtain closer association may have resulted from several influences. First the system of classification for seedlings and/or for adults may have been faulty. There is also the possibility that barley possesses an "adult plant" form of resistance, though the evidence is not at all conclusive. A low prevalence of rust on adult plants could have disturbed the association. Finally, contamination with other races of rust could have confused the results. In 1952 race 15B was very widespread over the entire upper Mississippi Valley and there were some indications that it was present in the nursery. However, race 15B has been shown to behave like the common races of stem rust and does not attack the varieties that carry the T factor for resistance (3).

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TRANSFER OF DWARF BUNT FROM WHEAT TO SLENDER WHEATGRASS AND FROM RYE TO WHEAT[1]

Jack P. Meiners[2]

The occurrence of dwarf bunt (Tilletia contraversa Kuehn) on wheat, rye, and certain grasses in the Pacific Northwest has raised the question of whether each of these hosts harbors its own pathogenically distinct race. Therefore, cross inoculation studies were undertaken to clarify this problem. Although it was reported previously (3) that wheat is susceptible to dwarf bunt from tall oatgrass and intermediate wheatgrass, the susceptibility of grasses to dwarf bunt from wheat has not been demonstrated by experiment.

In a recent series of inoculations slender wheatgrass (Agropyron trachycaulum (Link) Malte) became infected with dwarf bunt from wheat. Apparently this is the first report of a grass being found susceptible to wheat dwarf bunt by inoculation. The same inoculation procedure and inoculum also resulted in infection on Orin wheat.

The inoculation procedure was as follows: Dwarf bunt spores from Orin wheat collected near Pullman, Washington, were mixed with soil (Palouse silt loam) and the mixture placed in Petri dishes. The latter then were placed out-of-doors during mid-winter until spore germination (as evidenced by amount of germination on a microscope slide embedded in the soil) was secured. At this time the lid of the dish was removed, seed placed on the soil surface, the lid replaced, and the plates inverted. These were then incubated at 5°, 10°, and 15° C in refrigerating incubators until the coleoptiles of the seedlings were 1 to 2 inches long, after which they were transplanted to plant bands, placed outside to vernalize, and finally transplanted to the field in the spring.

Table 1. Percentages of dwarf bunt obtained in Primar slender wheatgrass and Orin wheat when inoculated with dwarf bunt from wheat.

Host Inoculated	Inoculation Temperature °C	Number Heads Total	Smutted	Percent Smut
Primar Slender Wheatgrass	5	175	9	5.1
"	10	225	0	0.0
"	15	200	0	0.0
Orin wheat	5	380	54	14.2
"	10	295	30	10.2
"	15	302	77	25.5
"	5	300	30	10.0
"	10	152	14	9.2
"	15	163	20	12.3

At 5° C, seed of slender wheatgrass germinated very slowly so that little germination had occurred even after 20 days, then the plate was transferred to the greenhouse (temperature 10-15° C), where better germination occurred.

Smut counts were made in August, 1955, and smut percentages were calculated on the basis of total and smutted heads.

The results are summarized in Table 1. In the case of Primar slender wheatgrass, dwarf

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bunt appeared only in the row in which the seed was incubated at 5° C for 20 days and then transferred to the greenhouse. The nine smutted heads were from two dwarf-bunted plants in this row. Higher infection percentages were obtained on Orin wheat (Table 1).

At the time the inoculations were made, slender wheatgrass had not been reported as a host for dwarf bunt. It was selected for this study because it has been found to be susceptible to Race T-16 (4), the dwarf bunt tester race of common bunt (2), and also because it heads well in one season. Its susceptibility to wheat dwarf bunt has since been established, however, in a planting of slender wheatgrass in naturally infested soil, near Worley, Idaho (5). In all cases of dwarf bunt observed on slender wheat grass the symptoms were generally similar to those of dwarf bunt on other grasses and on wheat. Infected culms were approximately half as tall as healthy ones. The symptoms on this host were illustrated in a previous report (5).

The circumstances under which dwarf bunt occurs on rye in the Pacific Northwest suggest a pathogenic relationship to the dwarf bunt on wheat. Experimental evidence now indicates this to be the case. Infection was obtained on one plant of the wheat variety, Hybrid 128, grown from seed inoculated with pregerminated spores of dwarf bunt from rye. This limited evidence needs further substantiation, especially in view of the negative results in cross inoculations with dwarf bunt from wheat and rye reported from Europe (1, 6).

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VERTICILLIUM WILT OF YELLOW-POPLAR

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In recent years a number of new hosts for Verticillium albo-atrum have been reported (3, 4). Only a few trees have been listed as hosts of this wilt; and of these, elm and maple are most frequently infected (1). A few years ago, in Milford, Connecticut, a yellow-poplar lawn tree (Liriodendron tulipifera L.) was reported by the owner to be gradually wilting and dying. An examination in September showed about 10 affected branches scattered through a 20-foot tree. Half of these branches had died during the summer and the rest had yellow wilting leaves with some dead lateral twigs. From greenish streaks in the wood of these branches, Verticillium was isolated.

In culture, the fungus first developed white mycelium with spore clusters resembling Cephalosporium. Later, verticillate conidiophores and microsclerotia indicated that the organism was Verticillium albo-atrum as described by Randolph (2). In monospore cultures, some isolates produced an abundance of conidia, but few microsclerotia. Other isolates showed an opposite reaction.

Cultures were maintained in the laboratory over winter on sterilized twigs of yellow-poplar and of elm. When transferred to malt agar medium, mycelial isolates from the yellow-poplar twigs consistently formed numerous microsclerotia, and those from the elm twigs formed very few. This tendency, however, was not maintained in isolates from monoconidia, which produced conidia and microsclerotia indiscriminately, without regard to the origin of the transfers.

Two series of inoculations in 2 consecutive years, totaling 42, were made in June in wounds in twigs of the current season's growth on young yellow-poplar nursery trees. Two sides of a triangular piece of bark were cut with a sterile scalpel. The piece of bark was turned back from the twig sufficiently to allow the insertion of a small piece of agar with mycelium and spores from a monospore culture. The bark was then pressed back against the inoculum and the inoculated part of the twig was wrapped with wet cotton and waxed paper. Eight check twigs were similarly treated, using sterile agar as the inoculum. The wrappings on all twigs were removed at the end of 1 week.

All inoculated twigs were collected the first of October each year, and were immediately cultured. All check wounds had healed. Many of the inoculated twigs had yellow wilting leaves, similar to those on the branches from which the Verticillium was originally isolated. Leaves on uninoculated trees were also beginning to turn yellow, but did not show the wilting that seemed to be a symptom of infection.

In 40 of the 42 inoculated twigs, greenish streaks were present in the wood, extending 1/2 inch to 4 inches from the inoculation wound. Dieback occurred in twigs with extensive discoloration. In 25 of the twigs, the streaks extended 1 inch or more beyond the wound; and from all of these, the Verticillium was reisolated. In microtome sections through the streaks, hyaline mycelium was found present in the vessels of the xylem in limited amount.

The results of the inoculations suggest that infection by Verticillium albo-atrum may cause wilting and death of young twigs of yellow-poplar. No previous report of yellow poplar as a host has been found.

The tree from which the Verticillium was originally isolated was growing in the lowest portion of a terraced lawn where the soil in the spring was frequently saturated with water that had drained down a hillside. This unfavorable situation may have weakened the tree sufficiently to predispose it to infection. At the time when the fungus was first isolated, all infected branches were pruned off close to the trunk and destroyed. During the 3 years since then, additional lateral branches, scattered throughout the tree, have gradually died. Each year, however, new growth has developed, and the disease has not seriously lessened the value of the tree as an ornamental.

The origin of the disease is not known. All other yellow-poplars in the vicinity were older and apparently healthy. No evidence of Verticillium in garden plants in the area or in maples or elms could be found. The occurrence of the disease is reported here to indicate that, under unfavorable growing conditions, branches of yellow-poplar may be killed by Verticillium albo-atrum.

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RECORDED DUTCH ELM DISEASE DISTRIBUTION IN NORTH AMERICA AS OF 1955[1]

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The most recently published map of Dutch elm disease distribution in the United States (1) shows whether the disease had been found in each State by 1952. More precise information gives a better appreciation of the large regions into which this disease apparently has not spread in the 26 years since it was first found in North America (2).

During the spring and again during the late fall of 1955, pathologists and entomologists in the affected States and Provinces were sent letters requesting isolation records for Graphium ulmi Schwarz, the fungus causing the Dutch elm disease. Records were requested for each town, where available; otherwise, for each county. The data in the replies to these letters, together with the culture records on file at the Shade Tree Laboratories, were used to prepare the accompanying map (Fig. 1).

That the Dutch elm disease was at some time found in a given town or county does not mean necessarily that it is now present in that locality. For example, G. ulmi was isolated from elms in several counties in Tennessee (1) in 1946 and 1948, but it was not found again in that State until 1955, when it was isolated in Knox County (3). Similarly, G. ulmi was found in Denver, Colorado, in 1948 (4) but it has not been found again in that State. Accordingly, Denver has been omitted from the present map, to permit the use of a larger scale.

Three, or possibly four, areas of infestation appear on the town- and county-level distribution map: (a) the Northeastern Seaboard States, with a center near New York City; (b) the St. Lawrence River valley in southern Quebec and eastern Ontario; and (c) the North Central States, with a center in Ohio, possibly including (d) the southernmost part of Ontario. These areas may be expected to merge soon.

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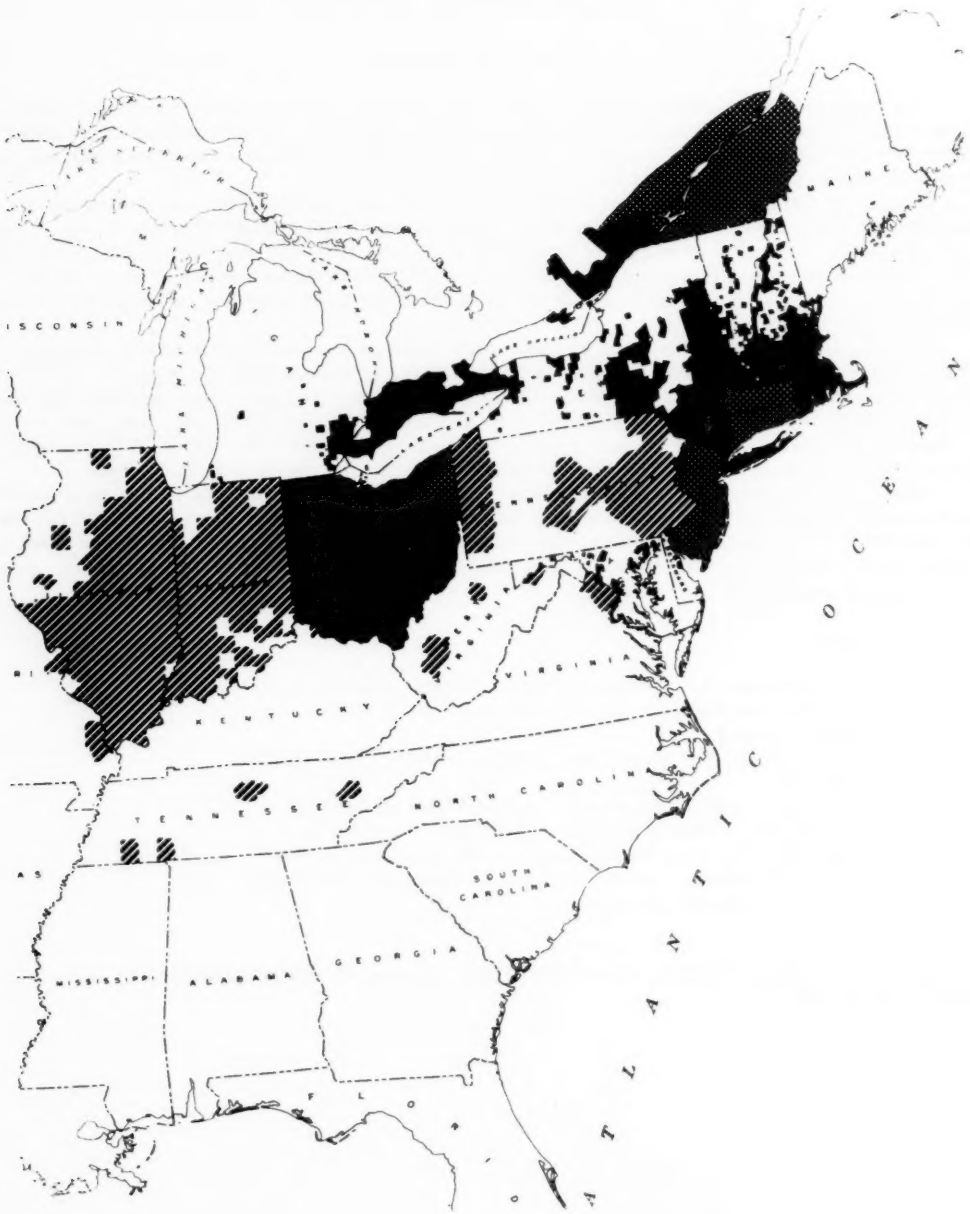
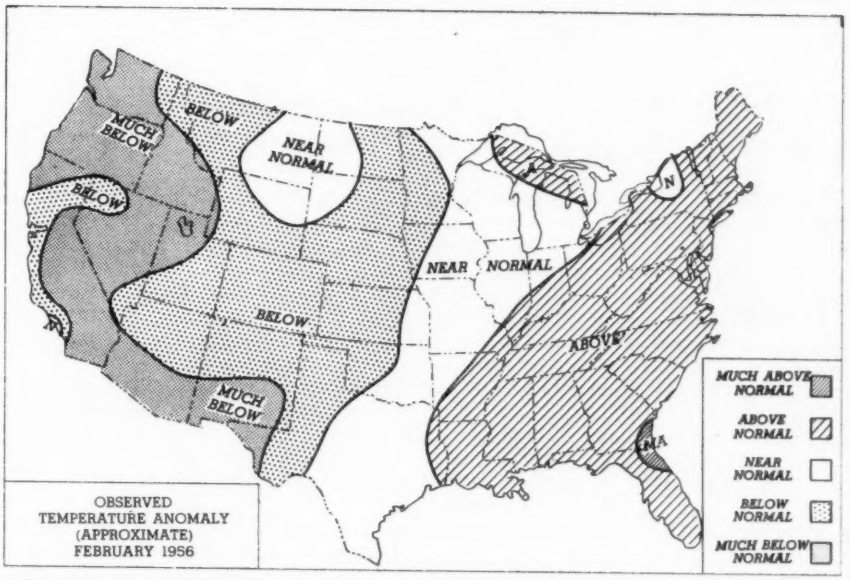
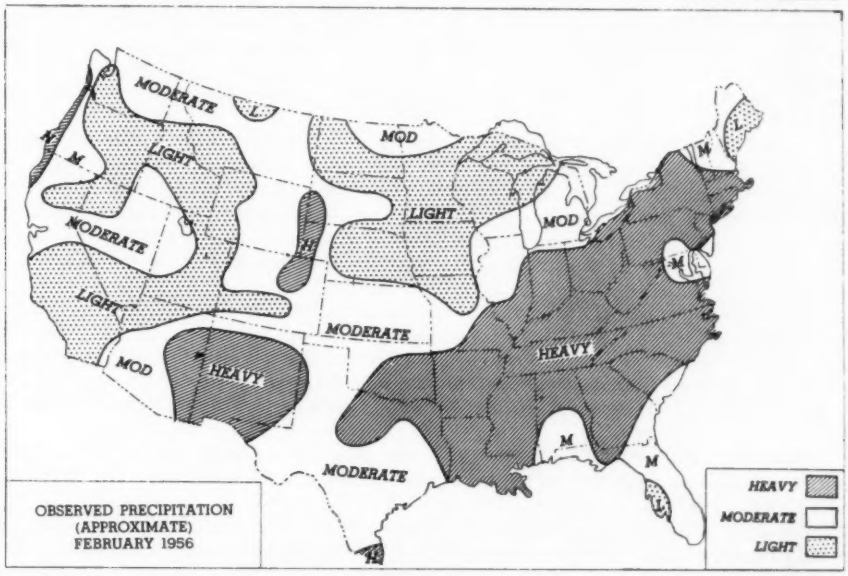


FIGURE 1. Dutch elm disease distribution in North America. Solid black areas indicate isolation records available for each town; diagonal lines indicate records available for each county (in Indiana the records are based on symptom observations as well as on isolations of the fungus); checkered areas indicate disease assumed to be throughout the state or region.





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.

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(See also PDR 35: 121-122; 36: 214, 494)

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